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An Address.[1]

AFTER SIXTY YEARS.

By GEORGE MONCRIEFF BARRON, O.B.E., M.B., Ch.M., F.R.A.C.S.,

President of the New South Wales Branch of the British Medical Association.

MAY I in the first place express my appreciation of the highest honour conferred by the medical profession in their electing me as President of the New South Wales Branch of the British Medical Association. When I look back at the names of illustrious men who have occupied the presidency, it is with feelings of humility that I accept this honoured position; and it is only the knowledge that I shall have the enthusiastic support of the members of our council as well as that of the capable and highly efficient staff that encourages me to endeavour to try to uphold the honourable traditions of our Association.

I should like on your behalf to express a deep appreciation of the services of our retiring President, Dr. B. T. Edye, who, though one of the hardest worked members of our profession, has carried out his arduous duties in a most capable manner through one of the most strenuous and exciting years in the history of our Association.

It is always a difficult problem to choose the subject for a presidential address, and as my predecessor dealt with the early pioneers of medicine in Australia, I have thought it opportune to carry on and to trace the history of our local association and to refer to some of the torch-bearers who have made our association the largest and most powerful Branch of the Parent Body.

In early days most of the medical men belonged to the medical section of the Royal Society of New South Wales. On February 9, 1880, a meeting of eight members was held. Mr. A. Moffitt was in

[1] Read at the annual meeting of the New South Wales Branch of the British Medical Association on March 30, 1939.

the chair, and Dr. F. Milford acted as honorary secretary when a motion was adopted: "That it is desirable that a branch of the British Medical Association be formed in New South Wales." A second meeting was held at the Cambridge Club Hotel on February 16, when draft by-laws were adopted. On March 1 officers and council were elected:

President: Arthur Renwick, M.D.
Vice-President: A. Moffitt, Esq.
Treasurer: H. G. A. Wright, Esq.
Honorary Secretary: F. Milford, M.D.
Council: G. Fortescue, M.B., H. N. Maclaurin, M.D., W. O'Reilly, M.D., C. K. MacKellar, M.B., G. Marshall, M.D., R. B. Shuetts, M.D.

The council in London on July 7, 1880, granted recognition to the formation of the branch.

It is interesting to look back and to discover who have occupied the position of President of our Association; consequently I have included such a list in this address. On perusing this list one can quite understand why the affairs of this Branch have been so well managed and why we are in the proud position we hold today. The list is as follows:

1880-1881	..	Arthur Renwick
1881-1882	..	Andrew Moffitt
1882-1883	..	George Fortescue
1883	..	Frederick Milford
1883-1884	..	Charles Kinnaird MacKellar
1884	..	Alfred Roberts
1884-1885	..	Frederick Harrison Quaife
1885-1886	..	Walter Joseph O'Reilly
1886-1887	..	Samuel Knaggs
1887-1888	..	John Mildred Creed
1888-1889	..	Thomas Chambers
1889-1890	..	Thomas Fiaschi
1890-1891	..	George Thomas Hankins
1891-1892	..	Robert Scot Skirving
1892-1893	..	John Mildred Creed
1893-1894	..	Ralph Worrall
1894-1895	..	William Henry Crago
1895-1896	..	Edward Johnstone Jenkins
1896-1897	..	Philip Sydney Jones
1897-1898	..	Charles Percy Barlee Clubbe
1898-1899	..	William Chisholm
1899-1900	..	Edward Thomas Thring
1900-1901	..	William Henry Coutie
1901-1902	..	Joseph Foreman
1902-1903	..	George Edward Rennie
1903-1904	..	Andrew John Brady.
1904-1905	..	Alexander MacCormick
1905-1906	..	Joseph Lieveston Beeston
1906-1907	..	Frank Antill Pockley.
1907-1908	..	Bernard James Newmarch
1908-1909	..	George Henry Abbott
1909-1910	..	Henry Vincent Critchley Hinder
1910-1911	..	James Adam Dick
1911-1912	..	Herbert Lethington Maitland
1912-1913	..	Clarence Read
1913-1915	..	David Thomas
1915-1916	..	George Armstrong
1916-1917	..	Sinclair Gillies
1917-1918	..	Robert Gordon Craig
1918-1919	..	Arthur Aubrey Palmer
1919-1920	..	Francis Percival Sandes
1920-1921	..	Charles Bickerton Blackburn
1921-1922	..	Fourness Barrington.
1922-1923	..	Thomas Walter Lipscomb
1923-1924	..	Charles Herbert Essery Lawes
1924-1925	..	Andrew Davidson.
1925-1926	..	Robert Blakeway Wade
1926-1927	..	Reginald Jeffrey Millard
1927-1928	..	Stewart Arthur Smith
1928-1929	..	James Eric Vernon Barling
1929-1930	..	Francis Brown Craig
1930	..	Esca Morris Humphery
1931-1932	..	George Bell
1932-1933	..	Alfred John Gibson
1933-1934	..	Alan Worsley Holmes à Court
1934-1935	..	Archibald John Collins
1935-1936	..	Arthur Madgwick Davidson
1936-1937	..	Edgar H. M. Stephen
1937-1938	..	Lindsay Alexander Dey
1938-1939	..	Benjamin Thomas Edye.

The early beginnings of the Branch and some very interesting biographies have been dealt with by Dr. R. Scot Skirving in a paper entitled "Fifty Years in a Changing World", published in The Medical Journal of Australia, July 23, 1932, and I would recommend all to read this illuminating article.

Sir Arthur Renwick, the first President, was one of the first three to take his B.A. degree in the University of Sydney, but he took his medical course at Edinburgh University. His life was a very full one as honorary medical officer of Sydney Hospital; founder of the Benevolent Asylum and Institute for Deaf, Dumb and Blind, and the State Children's Relief Department; President of the Medical Board for half his lifetime; senator of the university; dean of the faculty of medicine; a member of Parliament and Minister for Education. The Royal Hospital for Women at Paddington is the work of his genius and energy. For twenty-nine years he was President of Sydney Hospital. He was also associated with the old-age pension scheme, the original committee of which he was president.

Sir Arthur through a long life took the keenest interest in all movements for the moral welfare of the community and the advancement of true religion. There are few men to whom Sydney owes so much and who have manifested such devotion to community and national services. He was created a Knight Bachelor in 1892. Such a man was our first President. He died on November 22, 1908.

Sir Charles Kinnaird MacKellar was President in 1883-1884. He was a reformer, a pioneer and a leader. He was, while still a junior member, elected President of the British Medical Association. His restless, ambitious spirit, ever striving to effect what has been aptly termed "mass amelioration", ever determined to use his wide professional knowledge and ripe experience for the benefit of the whole community, compelled others to give him the first place—the place of leadership.

In 1882, when hygiene and preventive medicine were far less highly developed than they are now, he was appointed President of the Board of Health. He planned his department with the consummate skill of genius, and no one could have built up a better scheme for the Board of Health than he. He was responsible for the erection of the Coast Hospital at Little Bay. Today this has developed into one of our largest hospitals and is now being developed as the post-graduate school of medicine and surgery.

In 1902 he turned his attention to the care of delinquent and mentally deficient children, and was appointed President of the State Children's Relief Department; his work was outstanding in the reformation of the control of these children, and he placed the department in the high position it holds to this day. In England and America he was regarded as a pioneer in this branch of sociology. He was a man whose strong hand was ever extended to help others and to guide them.

He was created a Knight Bachelor in 1912 and in 1916 a Knight Commander of the Most Distinguished Order of Saint Michael and Saint George. He was a member of the Legislative Council and President of the Bank of New South Wales.

John Mildred Creed was President in 1887 and 1892. He was a resident medical officer at the Sydney Infirmary and later honorary surgeon to Sydney Hospital; he also acted as medical officer to an exploring expedition to the Northern Territory in 1867-1868.

He took a prominent part in the corporate life of the medical profession, and this at a time when associations and societies were not so well organized as they are today, when there was not the same stimulus as at present, but when the need was probably more apparent. He was secretary of our Branch and was the only man who has occupied the position of president on two occasions. He was always interested in affairs of State, and took an active part in parliamentary matters. He was a member for Upper Hunter in 1872, and became a member of the Legislative Council in 1885. He will be remembered most for his advocacy of cremation, and he was largely responsible for the introduction in 1886 of the bill to regulate it. He also played a prominent part in the legislation introduced in 1900 for the care and control of inebriates, and was an ardent advocate of the use of hypnotism in the treatment of chronic alcoholism.

Thomas H. Fiaschi was President in 1889. He graduated at Florence and Pisa, and practised at Cooktown, Queensland. He was a man of exceptional attainments and a pioneer who manifested courage in practice. Perhaps the most striking proof of his independence of thought at this stage was his introduction of the rectal route for the administration of quinine in the treatment of patients with malaria. In 1879 he moved to Windsor, New South Wales, where he turned his attention to surgery, and it soon became evident that his intimate knowledge of pathology and surgery, combined with great manual dexterity, placed him in the front rank of surgeons of his day. In 1894 he was appointed surgeon to Sydney Hospital; his colleagues were Dr. Goode, Dr. Chisholm and Dr. MacCormick.

He was an original member of the Association in 1880, and was early elected to the council and became President in 1889-1890. Those who knew him in earlier days remember his energy, his unflagging interest in his work, his solicitude for the welfare of all his patients no matter what their social standing, and his untiring efforts to gain fresh knowledge and impart it to his colleagues. His mind was an open one, and versatility was a strong characteristic in his professional and private life. He took part in the Italian and Abyssinian war in 1891, and when war broke out in South Africa in 1898 he was given command of Number 1 Field Hospital, and in 1914 Fiaschi was among the first to claim the right to serve his adopted country. He was placed in charge of Number 3 Australian General Hospital at Lemnos. His unflinching attitude to service in time of war seemed to some too stern and unyielding, but his discipline affected himself first of all and was always maintained for the benefit of the country and from a deep sense of duty. He worked very hard and achieved remarkable success. His interest in viticulture was well known. He was a well-informed student, who had mastered all the technical details of the wine industry.

Scot Skirving's panegyric can bear repetition here:

> Fiaschi was no ordinary man. His life proved it. He made good in no common way.
>
> He became a true and loyal son of the Empire and a great and useful colonist in this, one of its ultimate outposts. He rose to high professional distinction. Few, few indeed among his fellows had so wide and correct a knowledge of medicine as a whole. Learned, bold and enterprising, his keenness, which did not fade with years, was an example to us all, while his devotion to his profession and his determination to keep abreast of it were at once the admiration and despair of less strenuous souls. Physical and moral cowardice were alike unknown to him. Above all, he had a unique character and a great personality. He gave us an example of unspotted honour, of right professional conduct and of duty done at all costs in times of national stress.

Such was Fiaschi. It is always one of my most pleasant memories that I was privileged to be his house surgeon at Sydney Hospital, and many are the stories to be told of this beloved man's eccentricities.

William Henry Crago was President in 1894. He arrived in Sydney in 1865. He became a Licentiate of the Royal College of Physicians in 1883. He practised in William Street and later in College Street, and was honorary surgeon to the Royal Hospital for Women and to Sydney Hospital. His life's story might be written in the history of our Branch, as he was honorary treasurer for over forty years. This was merely one of the ways in which he devoted himself to its service. As honorary treasurer he had a great deal to do with the erection of the B.M.A. Building in Elizabeth Street, and the success of the venture was in no small way due to him; later he was prime mover in the erection of our present handsome building. The Branch recognized the debt that it owed to Crago, and showed its appreciation by electing him a vice-president, an honour that had not previously been granted to any member of the Branch. A portrait painted by Sir John Longstaff, R.A., was presented to him, and now hangs on the wall of the council chamber which bears his name. In 1929

the gold medal of the British Medical Association in Australia was presented to him.

Those who do most for medicine and for the whole body of medical practitioners are not always in the public eye, nor do they receive the recognition that by right is theirs. Some of the effective workers are unobtrusive; they do their good works almost by stealth, and only those in close contact with them behind the scenes know the true value of their deeds and the extent of their devotion and self-sacrifice. The younger graduates in New South Wales knew Crago as a quiet, diffident honorary treasurer. They saw he was treated with deference and they could not do other than appreciate the long years of service that he had given to the Association; but it is doubtful whether they knew how much they really owed to him.

Sir Phillip Sydney Jones was President in 1896. He was appointed to the Sydney Infirmary in 1861. In 1887 he was appointed honorary consulting physician to the Royal Prince Alfred Hospital and continued in this capacity until his death in 1918. In 1867 he joined the Royal Society of New South Wales and took a leading part in the medical section of that body. In 1882 he was a member of the Royal Commission appointed to investigate and to report on the arrangements of the quarantine station; the result was the establishment of the quarantine station at North Head for the reception of all persons suffering from infectious disease who came to New South Wales by sea. He was a member of the university senate from 1887 until his death. For thirty years he was a member of the Medical Board.

He will be remembered for his unceasing efforts to stamp out tuberculosis. He was a pioneer of open-air treatment, and was instrumental in the establishment of the Queen Victoria Homes at Thirlmere and Wentworth Falls. He took a leading part in the foundation of the National Association for the Prevention and Cure of Consumption, and gave much valuable advice concerning the institution of anti-tuberculosis dispensaries which have been founded in New South Wales. He took an active part in many charitable institutions, such as the Institute for the Deaf, Dumb and Blind. He was a member of the Linnean Society and trustee of the Australian Museum, and he was associated with the work of the Kindergarten Union. He gave to the medical profession in New South Wales the pattern of the wise physician of exemplary probity, of unfailing courtesy and of widest charity. He utilized his professional attainments as far as possible for the benefit of his fellow citizens, and employed his great experience in the care and treatment of the sick for the healing of those who were ignorant whence cure came. His advocacy of better methods for the control of tuberculosis reveals to us the noble spirit of the learned and skilled physician.

For a quarter of a century he was recognized as the leader of the medical profession in New South Wales.

Sir Charles P. B. Clubbe was President in 1897. He was honorary surgeon to the Royal Prince Alfred Hospital from 1889 to 1907. He was lecturer in clinical surgery, and I was fortunate enough to be one of his students. When he gave a lecture demonstration the subject was presented in such a way that the student felt he would never make a wrong diagnosis of the condition demonstrated. It was the Children's Hospital, now the Royal Alexandra Hospital for Children, which was dearest to Clubbe's heart, and his name will ever be associated with it. In the early days he was its mainstay; later he became its sponsor and largely controlled its destiny. (He was a member of the staff for forty-eight years and president for twenty-eight years.) His was the imagination which saw the need for extension and rebuilding; he always saw that the advancement was made before the sheer weight of public opinion compelled an advance. The organization of the Infantile Paralysis Committee was one of his most successful undertakings.

His concern in the poliomyelitis problem was real, and several articles from his pen were published. The Bush Nursing Association was another of his activities. He was trustee and chairman of the District Nursing Association, and he helped to organize the baby clinics. His book on the diagnosis and treatment of intussusception is a classic to this day.

George Edward Rennie was President in 1902. He was born at Sydney in 1861. He graduated at London University College with highest honours, and became a Fellow of the Royal College of Physicians in 1907. He was on the staff of the Royal Prince Alfred Hospital as pathologist and physician. He turned his attention to neurology and was for many years lecturer in clinical medicine. Those of us who were fortunate enough to have been in his classes will always remember with gratitude and pleasure his devotion to his work. He was an inspiration to excel; he had a brilliant intellect; he was a devoted student of his art, a methodical clinician and an intensely conscientious and loyal servant to his patients and to the medical service with which he was connected. He was the best systematic teacher to whom I ever listened; his lectures showed in their delivery the hours spent on their preparation and a unique power of coordination and systematic presentation; thus they revealed the master mind.

He was an active member of our council and in 1901 was appointed editor of the *Australasian Medical Gazette*, which he edited with eminent success for over twelve and a half years. The volumes of this gazette stand as a monument of his industry, ability and efficiency.

Andrew John Brady was President in 1903. He was an original member in 1880. He became the first ear, nose and throat specialist in New South Wales, and he created the ear, nose and throat department at Sydney Hospital in 1887. He was also invited to occupy that specialty at the Royal Prince Alfred

Hospital, and accepted. At this time there was no training school for nurses and he pressed for this instrument to raise the standard of nursing in Australia. While he was working quietly but with the expenditure of much energy at his own specialty his precision led him to be the prime mover in the establishment of many large and small reforms in medical work. From 1889 until 1923 he was a member of the council. Not only did he take an active and prominent part in medico-political activities of the Branch; he was also keenly interested in the various sessions of the Australasian medical congresses. He contributed to the literature of his specialty, and his articles were always instructive and important.

Apart from his skill and knowledge and from his unswerving determination to achieve his objectives, he was a courtly gentleman, lovable, sensitive to the suffering of humanity, gentle and large-hearted. His reputation was world-wide.

Sydney Hospital is indebted to him for more than the mere service over a period of fifty-three years.

Robert Gordon Craig was President in 1917. He graduated at the University of Sydney in 1894. He was honorary surgeon to the Royal Prince Alfred Hospital. He was keenly interested in genito-urinary surgery, and founded the department of urology at the Royal Prince Alfred Hospital by a gift of £20,000, with the object of the furtherance of urological research and the appointment of fellows in urology. He was a foundation member of the Royal Australasian College of Surgeons and made provision for the college in his will; it benefited to the extent of £82,000.

He was recognized as a conscientious and reliable surgeon; his fame has spread beyond our continent. He not only made a name for himself, but also did much for surgery. He did what few medical practitioners do: he gave back to the world of medicine some of the tangible wealth that he had won, so that the younger men might find the path easier than he had found it. He was for many years a member of the council of the New South Wales Branch. He undoubtedly left his mark in the world of medicine and a name which will be honoured.

Robert Henry Todd.

No record of the history of the Branch would be complete without mention of the work done by Dr. Robert Henry Todd. He was born in London in 1859, took his arts course at Oxford, studied medicine in Dublin, and arrived in Sydney in 1887. He was honorary anæsthetist at Prince Alfred Hospital, where he did some useful work in organizing this department. Later he practised at Maclean, on the Clarence River. Here he suffered from an infection of his wrist, which prevented him from carrying out the routine of general practice. As a result he studied law and was called to the Bar of New South Wales in 1894. He was soon appointed city coroner and was also chairman of several industrial boards. He was peculiarly well suited for these duties. In 1904 he was largely responsible for the drawing up of the report of the Royal Commission on the decline of the birth rate in New South Wales.

In 1891 his first connexion was made with the British Medical Association. Five years later he was elected a member of the Branch council. In 1908 he was chosen as honorary secretary of the Branch. He served in this position until he retired in 1931. History shows that advance in any walk of life has been achieved by the efforts of one or more persons who set up an ideal, who made themselves of no account and strove valiantly to attain their objective. Medicine has been served in this way by not a few. The history of our Association holds a record of service and devotion by those who believed that the altar of medicine should be served by men, clean of heart and hand, prepared to offer sacrifice even of themselves.

To say that Todd was honorary secretary to the New South Wales Branch would not convey to an outsider any adequate idea of his activities. His capacity for work was enormous and this, combined with his ethical outlook, his idealism and the caution of his legal mind, had the only possible result in the progress of the Branch and of the Association in Australia along every avenue.

His organization of the affairs of the Branch was along sound lines. As the membership grew he realized that the council could not possibly deal with the minutiæ of every question or dispute requiring consideration, and he saw that the large body of members would wish to have a louder voice in Association affairs than was possible when the council had no machinery for consulting the members whenever it desired. As a result the work of the council was split up among committees and the local associations of members were brought into being. These associations acted in a most useful way as a liaison between the council and members of the Branch in a district.

The members of the local associations were brought into close relationship with the council by an annual conference of delegates with the council.

The problems of friendly society lodge practice exercised Todd's mind, and the smooth working of the model lodge agreement is in no small measure due to his care and organizing ability.

Todd's activities were not confined to the New South Wales Branch. He was one of those who took steps to form the Federal Committee of the British Medical Association in Australia, with the object of coordinating the work of the six Australian Branches. He was responsible for the drafting of the constitution of the Federal Committee. In 1918 he became honorary secretary of the committee and held the position until the time of his death. In conducting the affairs of the Association he was meticulous in his attention to detail. His legal training helped him to foresee difficulties, and he could generally suggest a way of overcoming them.

In 1922 he received the signal honour at the hands of the Council of the Parent Body of election to the position of Vice-President of the Association.

When he retired from the honorary secretaryship of the New South Wales Branch in 1931 the members of the Branch offered to Todd the position of president-elect. It was the highest honour they had to offer him. Had he succeeded to the position of president he would have occupied the chair which was presented by him to the Branch in 1911. In the designing and construction of this chair he had displayed much taste and had taken infinite care.

Todd's activities outside the sphere of the British Medical Association were numerous. He led a full life, spared not himself, but gave freely of all that was within him. He was one of what is becoming known as "the old school", a type that is said to be dying out. Were there a man in every medical community, a man actuated by his beliefs, possessed of his courtesy and endowed with his qualities, the world of medicine would be more wholesome, there would be less commercialism, and there would be more brotherly love.

The hall we are in tonight has been dedicated to his memory as the Robert H. Todd Assembly Hall.

Other Prominent Members.

Such were the activities of some of the more prominent members who guided the affairs of our association during the past sixty years, and time will not permit me to dwell upon the doings of many others of whom I could speak; but the opportunity to mention the work done by such men as our friend Dr. Ralph Worrall, who is still with us, cannot be passed by, as he was one of the stalwarts who built up our Branch in the early days, he having occupied the chair in 1893.

Another notable personality is Dr. Robert Scot Skirving, who bore much of the brunt of the arduous work of secretary *et cetera* in the early days and who did such great work in directing affairs and in contributing in so many ways by both his facile pen and silvery tongue, for he has always been one of the greatest personalities in our profession. Many of us who had the opportunity of attending his clinical lectures in medicine at the Royal Prince Alfred Hospital retain the happiest memories of some of the most delightful and entertaining afternoons of our medical course, when our lecturer paced up and down the strip of carpet alongside the bed of the patient being lectured upon. Any lecture or after-dinner speech by Scot Skirving was always the star piece of the evening, and it is a tragedy that we do not see more of him these days. My excuse for speaking thus is expressed in the following lines of a poem:

If with pleasure you are viewing any work a man is doing,
If you like him or you love him, tell him now;
Don't withhold your approbation 'til the parson makes oration
And he lies with snowy lilies o'er his brow;
For no matter how you shout it, he won't really care about it;
He won't know how many teardrops you have shed;
If you think some praise is due him, now's the time to slip it to him,
For he cannot read his tombstone when he's dead.

The meetings of the Branch in the beginning were held at the rooms of the Royal Society, and in 1911 a block of land in Elizabeth Street, near Hunter Street, was bought and a building was erected, which was occupied by us until it was resumed for the widening of Elizabeth Street. Then our present building here at 135, Macquarie Street was built, the foundation stone being laid by Sir Ewen Maclean on September 7, 1929. The building was opened by Sir Philip Game, then Governor of New South Wales, on October 3, 1930, and today it is one of the most beautiful buildings in our city.

The Ethics of the Medical Profession.

During my term of office as a member of this council I have been associated with the Ethics Committee, and perhaps it will not be out of place if I make a few remarks upon this side of our work; for a great many questions could easily be settled without any reference to this committee if a little forbearance and understanding were exhibited by members one toward the other.

One may ask what is medical ethics? A simple and literal definition might be given as a practical observance in the daily walk of professional life of the divine command: "Whatsoever ye would that men should do unto you, even so do ye also unto them." And here I would suggest that young practitioners, and perhaps older ones, should make a careful study of the "Handbook for Qualified Medical Practitioners", which was recently published by this Branch and which is presented to all graduates in medicine. This small book is full of valuable information; and if all the advice which is offered were absorbed and practised, there would be much less work for our ethics committee to do. The Hippocratic oath, which we have all accepted, might be read once more, and if its precepts were carried out as recommended, our profession would be a much happier one today.

Medical ethics, to be effective, must be based on the principles of religion and morality, and must embody the reciprocal duties and rights of the profession and the public.

Medical practitioners, as conservators of the public health, are bound to bear emphatic testimony against quackery in all its forms, whether it appears with its usual effrontery or masks itself under the plausible garb of philanthropy or the ostensible cloak of religion.

Professional differences not infrequently arise from want of candour on the part of patients, their relatives or attendant friends, and probably constitute the most fruitful source of the unhappy heart burnings and jealousies which so frequently disgrace our profession. A medical man cannot, therefore, be too cautious how he receives and acts upon invidious statements said to have been made in reference to a case by a professional brother or other person. Such reputed remarks are often either so misunderstood and misrepresented or so wilfully perverted as to give rise to serious disputes and lasting estrangements, which a personal interview

or a mutually courteous note of inquiry and explanation would prevent or remove. Be it therefore a special obligation on the faculty to be ever careful and wary in respect to statements which involve disparagement of a brother practitioner.

The faculty is under the strongest ethical obligations to maintain and justify the character which has been awarded by the most learned men to the medical profession, for general and extensive knowledge, liberality and dignity of sentiment and great beneficence. To uphold so honourable a position it cannot be too strongly impressed upon one and all that it is essentially necessary that a medical practitioner should be an educated, cultured and right-minded Christian gentleman, and that he should fulfil his duties with kindness, with courtesy and with a just sense of true dignity, engendered of self-respect. It is the duty of a practitioner to himself, as well as to those entrusted to his care, to cultivate the spirit of kindness, gentleness and thoughtfulness, to be suave, courteous and cheerful, especially at the couch of the sick, and also to be scrupulously neat in person and appearance.

There is no profession from the members of which greater purity of character and a higher standard of moral excellence are required than the medical profession; and to attain such eminence is a duty which every practitioner owes alike to his profession and to his patients.

The calling of a medical practitioner differs from other learned professions in that the practitioner deals with the lives of men and women. He is present at birth and maybe closes their eyes in death. In the interval between life and death he is with them in sickness and in health and is made the sacred repository of their inmost secrets. He is thus the most trusted of all men, and his responsibilities are as great as the trust reposed in him. In these circumstances it is necessary that his actions should be governed by the most rigid code of ethics and that he should have some court of appeal when he is in doubt as to what course of action he should follow. These requirements are filled by the British Medical Association.

The objects of the Association are the promotion of medical and allied sciences and the maintenance of the honour and the interests of the medical profession.

The Association is not a statutory body and it has no legal control over its members. It is a voluntary body, and if any member is adjudged guilty of having acted in a manner calculated to bring the profession into disrepute, he may be deprived of membership of the Association. On the other hand, the Medical Board of New South Wales is a statutory body and alone has the power to deregister a medical practitioner. This is not always understood by lay people. The honour of the profession is maintained by our code of ethics. This code covers not only the relationship of the practitioner with his patient, but such questions as advertising, canvassing for a practice, the splitting of fees, the acceptance of commissions, the treatment of a patient who is under the care of another practitioner, the association with unregistered practitioners, and so forth.

The Association also acts for medical practitioners in all matters of medico-political significance. It endeavours to secure proper conditions for friendly society practice, other contract practice and for departmental and other services. Emphasis may be placed on the feeling of brotherly love that inevitably results from the association of persons striving for a common object. The object common to all medical practitioners is the prevention of disease.

There should therefore be no hesitation on the part of new graduates to join the British Medical Association.

It will be noted that in the modern conception of medical practice great emphasis is placed on the role of the general practitioner as health adviser. Owing to the advances of scientific knowledge many specialisms have developed, and there is no reason to suppose that this movement will be less rapid in the future than it has been in recent times. According to Herodotus, "The Father of History", who travelled in Egypt in the fourth century B.C., medicine had developed to a degree of specialism not equalled even now. He avers that there were doctors who exclusively treated diseases of one part of the body, as the great toe, the little finger *et cetera*. On the other hand there has emerged a growing public appreciation of the value of health and of the importance of early attention to departures from normal and a demand for the services of the general practitioner as health adviser.

This movement is sure to develop, and the national policy for the promotion of health should be so framed as to encourage it. It appears that the role of health adviser undertaken by the general practitioner is a national development of ordinary medical practice, and that the training of the student of medicine should be adapted to fit him for it.

It is of primary importance that the organization of a national health service should be based upon the family as the normal unit and on the general practitioner as the normal medical attendant. The first essential for the proper and efficient treatment of individual persons is therefore not institutional, but personal service, such as can be rendered in their own homes only by a general practitioner who has the continuous care of their health, who will afford them such professional services as he can render personally, and who will make it his duty to see that they obtain full advantage of all the further auxiliary services that may be otherwise provided.

"The Medical Journal of Australia."

Some misunderstanding exists in connexion with the management of our medical journal. Originally the *Australasian Medical Gazette* (New South Wales) was published by a private individual, but was purchased through the enthusiasm of four men (Rennie, Huxtable, Crago and Worrall). Dr. Crago

acted as manager from 1895 to 1914. Later a scheme was devised for a Federal medical journal; the Victorian Branch handed over its *Australian Medical Journal* and the New South Wales Branch handed over its *Australasian Medical Gazette*, both without remuneration.

The Australasian Medical Publishing Company, Limited, was formed in 1913 with no share capital, but with money raised by debentures from medical practitioners in the several States, and this company produced the first issue of The Medical Journal of Australia on July 4, 1914. The company is thus owned by and exists for the profession, and any profits made will eventually go to the Branches.

The journal is not the official organ of the British Medical Association, and your council does not control its affairs, nor is it responsible for any articles or opinions published by its Editor or contributors; but for authoritative information on Federal matters the journal looks to the Federal Council, and for purely intrastate matters to the individual Branch councils.

Conclusion.

The past year has been one of great anxiety to your council. Regarding the question of national insurance, so much has been said and written on this subject that I shall not belabour the matter here. I would merely state that in the past, when we passed through such a crisis as in the lodge question, the members were wholeheartedly behind your council, and I trust that the same will hold good in our dealing with national insurance.

Your council will see that the interests of our profession are properly safeguarded before we recommend any acquiescence in accepting terms and regulations offered by the Government.

Another question causing anxiety is the overcrowding of the medical profession today. Parents would be well advised to look ahead and to consider the matter seriously before they start their sons and daughters on a career in medicine when the outlook is so gloomy as it is today. At present in New South Wales 2,296 medical practitioners are practising, and there are 900 medical students; with a population of 2,724,657, the recognized quota is to be very considerably lowered, and as a consequence the danger of inferior service to the public is one which is to be deplored.

It has always been our proud boast that we maintain the high standard of the profession ethically, scientifically and from the standpoint of national welfare, and we are able to hold before us such high ideals when we consider the lives of those who held office in our Association during the past sixty years, which I have related to you here tonight.

The memories of these lives and the associations we have had in our council here recall to me the words of the poet:

> Long, long be my heart with such memories filled;
> Like the vase in which roses have once been distilled—
> You may break, you may shatter the vase, if you will
> But the scent of the roses will hang round it still.

THE ÆTIOLOGY OF CHRONIC NEPHRITIS IN CHILDHOOD.

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The nomenclature of Bright's disease has become so confused and complicated owing to our efforts to find terms to suit the clinician, the biochemist and the pathologist, that it is a wise procedure in any essay on nephritis to stipulate at the outset what is meant by the title chosen. Even in the subdivision of chronic nephritis there is schism, for two entirely different lesions are given this name, "chronic interstitial nephritis" and "chronic parenchymatous nephritis". To simplify matters, the latter has been excluded from this article. The word interstitial has been omitted because the lesions are not confined to the interstitial tissue.

Chronic nephritis is a term used perhaps more frequently in pathology than at the bedside. It has its clinical and biochemical significance, however. Clinically it includes all those patients presenting the picture of chronic renal failure or insufficiency, such as thirst, polyuria, frequency of micturition, headache, vomiting, anorexia, pallor and failure of growth, to which are added the manifestations of uræmia. The urine is pale, of low specific gravity, and contains a faint cloud of albumin. The biochemist reports grossly impaired urea concentration of the urine; and examination of the blood reveals retention of non-protein nitrogen, raised blood phosphorus and lowered blood calcium content.

Chronic nephritis is not synonymous with renal fibrosis; for that may be confined to one small part of a kidney and be of no clinical significance. When the fibrosis is of such an extent as to interfere with kidney function and eventually to give the picture of chronic renal insufficiency, or even to cause death before this stage is reached, then may we designate it chronic nephritis.

Review of Literature.

No doubt the confusion in the nomenclature, as well as the comparative rarity of the condition in the child, explains the paucity of the local literature on this disease.

In 1927 Grieve[14] presented to the Melbourne Pædiatric Society a boy, aged nine years, who was suffering from chronic nephritis, and when discussing the ætiology he mentioned a congenital dilatation of the ureters as one causative factor. In 1929 Nye[30] showed that chronic nephritis was more common amongst children in Queensland than in any of the other Australian States. He was not satisfied by the explanation that scarlet fever and acute nephritis were more frequently met with in this State, and produced evidence to show that lead poisoning was a potent factor in this high incidence. Maddox[26] in a very instructive article came to the conclusion that the ætiology was prob-

ably congenital, either an agenesia, abiotrophy or an intrauterine nephritis. In a small series of cases he was unable to find any evidence of renal damage to the child following on the toxæmias of pregnancy. The communication of Russell[41] dealt with the treatment of this disease.

There have, however, been numerous communications in overseas journals on the subject since Gull and Sutton,[16] in 1872, in their celebrated article on "arterio-capillary fibrosis", described, amongst others, a girl aged nine years, who had very small, irregular, contracted kidneys. W. H. Barlow[3] in 1884 described a case of "cirrhosis of the kidney" in a child aged six years. Lucas[25] in 1883 observed that "the phenomenon of late rickets and albuminuria are too frequently connected to be matters of chance". Guthrie[17] in 1900 reported the disease in a girl, aged seven years, and during the same year Sequeira[42] reported a death from uræmia of a boy, aged three years, whose main symptom was polyuria. Milligan[28] in 1902 described a case of chronic interstitial nephritis in a boy aged seven years. Fletcher[12] in 1911 clearly defined the ætiological connexion between chronic kidney disease and bony deformities. In the first decade of the present century Parsons[31] became interested in this association of infantilism and chronic kidney disease, and since then he has contributed largely to the literature. His first article appeared in 1911, and with Miller[32] in 1912 he described a girl, aged six years and six months, suffering from "drinking diabetes". The kidneys *post mortem* were extremely small and granular. Naish[29] was struck by the association of infantilism and chronic interstitial nephritis in the case he described in 1912. Paterson[34] presented three cases of renal dwarfism before the Royal Society of Medicine in 1920. Barber,[2] in a striking paper on the same subject, defined it as a "condition of stunted development, associated with bone deformities of the 'late rickets' type due to an insidious chronic interstitial nephritis of obscure ætiology"; he presented several cases to bear this out.

Greene[13] in 1922 reported a case of chronic nephritis and gave an exhaustive review of the literature up to that date. In a discussion of the ætiology he admitted that cases of acute nephritis might progress to the chronic state; but in the great majority of cases he concluded that the origin was congenital, due either to a developmental defect or to an infection or toxæmia of the fœtus *in utero* in association with kidney disease in the mother.

In 1928 Spence,[44] in an illuminating article, not only described detailed reports of a number of cases, but made an effort to classify them on an ætiological basis. He described three groups: chronic parenchymatous nephritis, chronic interstitial nephritis and chronic ascending pyelonephritis. Chronic interstitial nephritis was subdivided firstly into cases without previous history of acute nephritis or preceding sepsis, secondly cases with such a history, and thirdly cases due to arteriosclerosis.

Previous to Greene's and Spence's articles there were a few sporadic attempts to explain the ætiology, notably by Guthrie,[18] in 1897, and Sawyer,[40] in 1906. Guthrie,[18] after giving an excellent description of the signs and symptoms of chronic nephritis, attempted to explain the ætiology as: (i) scarlatinal; (ii) previous acute interstitial nephritis, the cause of which was difficult to decide, but chilling probably being a factor; and (iii) ascending infection due to a previous bladder trouble or obstruction of the ureters or exit from the bladder. He noticed the hydronephrosis and hydroureters in these cases and gave an ingenious explanation. He suggested that owing to the great polyuria of the granular kidneys the bladder became over-distended, and this over-distension was responsible for back pressure on the ureters and kidneys. The more granular the kidney, the greater the polyuria for that kidney, and so the greater the degree of hydroureters and hydronephrosis.

Sawyer[40] also favoured the vicious circle: obstruction, hydronephrosis and hydroureters, granular kidney, polyuria. He stated that "syphilis either congenital or acquired may be the chief factor in the causation of the chronic interstitial nephritis of childhood, and may play a greater part in the ætiology of the disease in adults than is usually attributed to it". He thought there was just a possibility that the disease in children might be the sequela of acute congenital or even of an intrauterine nephritis, but felt that it was rarely, if ever, a further stage of the large white kidney.

Boyd[4] in 1929 preferred to divide the cases into two groups: (i) a first group in which the signs and symptoms followed an acute nephritis, and (ii) a second group in which there were no previous symptoms to direct attention to the kidneys until chronic changes were apparent. The underlying cause in the second group is not made clear.

Calvin and Rosenblum[6] in 1920 reported two cases of what they regard as an unusual combination of advanced renal insufficiency, retention of non-protein nitrogen and a normal blood pressure. Ellis and Evans[8] in 1933 investigated twenty cases of renal dwarfism. In eleven out of fourteen in which recordings of the blood pressure were taken, it was found to be within normal limits. Because of the consistent finding in the urinary tract of dilatation from the neck of the bladder upwards, and also because of the absence of obvious organic lesions, the ætiology suggested was a neuromuscular defect at the junction of the bladder and the urethra. They found the condition *post mortem* in infants, aged three weeks and six months respectively, and assumed that it was probably congenital. Hypertension, they considered, was absent only in cases associated with dilatation of the urinary tract, and the contracted kidney of true insidious nephritis was accompanied, in children as in adults, by arterial hypertension.

V. H. Ellis[9] in 1933 described two most unusual cases of renal rickets following acute nephritis in previously healthy children, in one case after six

years and in the other after seven years. He admitted, however, the difficulty of proof in any individual case that the kidneys were healthy at the onset of the acquired disease.

Ætiological Considerations.

Owing to the fact that chronic nephritis is the final stage of a chronic process, the beginnings of which are usually wrapped in mystery, the ætiology has never been, and probably never will be, completely disclosed. It is just as difficult to tell the origin of a scarred granular kidney as of a scar anywhere else in the body, and especially difficult since the kidney is such a deep-seated organ. In children the task is made perhaps less difficult in that mixed types are not so frequently met with as in adults.

Congenital Abnormalities.

When such conditions as hydronephrosis and hydroureter, stenosis of ureter, renal aplasia, urethral valve, neuro-muscular defect *et cetera* occur in children, it is almost certain, though not absolutely so, that the condition is congenital in origin. The shrivelled fibrotic small kidneys first described by Rose Bradford[5] have a more obscure origin. It would seem that these too are congenital, being due to a renal fibrosis commencing in intra-uterine life. It is difficult to imagine that such a process could insidiously take place during life without making its presence felt in some way or other.

Spence[44] found the commonest type that without previous history of acute or subacute nephritis, with advancing azotæmia and with little or no rise in the blood pressure. He added that "in many of these the history suggests that the symptoms of the disease started in the first year of life". He therefore favoured the congenital theory, but thought that this was probably not the underlying factor in all instances. It did not adequately explain the cases described by Barber,[2] which occurred early in adolescence, and by Bradford,[5] which occurred in young adults dying in uræmia, with small white kidneys, without cardiac hypertrophy or arterio-sclerosis. But does the fact that they make their presence felt in adolescence or even in adult life preclude a congenital origin? How frequently is congenital cystic disease of the kidney diagnosed only when the patient has passed his childhood years? Greene[13] has come to the conclusion that in the great majority of cases the origin is congenital.

Fletcher[12] expressed the opinion, when discussing his interesting case, that the disease dated from intrauterine existence. Since that time many others have concurred in this conception, and in fact no other reasonable explanation would appear to be forthcoming.

Symptoms dating from birth in many cases and lesions found *post mortem* at a very early age also favour the theory of congenital origin of such conditions. King and Sheldon's[22] patient died at three weeks; so did one of Ellis and Evans's[8] patients.

Cases also have been described in which there were associated congenital abnormalities elsewhere. In the case described by Weigert[46] the patient had a congenital malformation of the heart.

It is at least certain that these abnormalities may, if the fibrosis and scarring progress beyond a certain stage or if further burdens are thrown upon the kidneys, give a clinical picture of chronic nephritis; and indeed on histological examination the kidneys are often difficult or impossible to distinguish from those of chronic nephritis arising in any other way.

End-Result of Acute Infection.

Most authorities, however, agree now that chronic nephritis may be the terminal stage of acute or subacute nephritis; but even this termination has been held in doubt for a long time. J. E. Sawyer[40] in *St. Thomas's Hospital Reports* says:

> In my opinion the contracted granular kidney rarely if ever is a further stage of the large white kidney, but is a result of a primary growth or proliferation of the interlobular connective tissue.

Aldrich[1] followed up over periods of one to ten years 99 patients who had suffered from acute nephritis. He was unable to find any case of chronic nephritis following on the acute type. Of 24 patients with chronic nephritis, two gave a previous history of acute nephritis. He did not regard this evidence as significant.

L. W. Hill,[19][20][21] however, in a series of articles on nephritis furnished convincing evidence that this result could follow; and in 1921 James[7] reported sixty-seven cases of acute nephritis, nine of which terminated in chronic disease of the kidneys. Tallermann[45] followed up twenty-seven patients who suffered from acute nephritis. None went on to chronic nephritis, but the longest period of observation was only three years and six months. Four patients showed evidence of persisting renal damage after this time, but their general health was good. It is probable that if these children were subjected to one or two more attacks of acute nephritis, the likelihood of their terminating as chronic renal failure would be much enhanced.

J. D. Mann[27] as long ago as 1895 recorded a case in which a patient under his care for the long period of twenty-eight years began her illness with an acute nephritis complicating scarlet fever, from which recovery ensued, only to be followed by attacks of subacute nephritis; the illness terminated with the signs and symptoms of chronic nephritis. At autopsy this was verified.

Spence[44] was of the opinion that this method of termination did take place, and quoted a case to support such a contention.

It is obvious from the doubt which was originally cast upon the occurrence of this event, and from the small number of cases which have been described, that this termination is not a common one. Its comparative frequency of recent years is due to the fact that it is more carefully looked for. The efforts of a certain school to change our conception of nephritis from a confusing variety of distinct diseases to a single continuous process passing

through stages recognizable clinically and pathologically have also assisted in this direction.

Malignant Hypertension.

As malignant hypertension is a disease which attacks the intimate vasculature, in which the kidney is rich, it is not surprising that some cases of chronic nephritis are traceable to this source. The dearth of reported cases, however, is an indication of the rarity of hypertension presenting the clinical picture of chronic nephritis, though no doubt pathological evidence of the diseases exists in practically every instance. The difficulty of establishing in any one case whether the hypertension or the chronic nephritis comes first is practically insurmountable.

Evans[10] describes the condition of a boy, aged seven years, which is probably of hypertensive origin. Spence's[44] case in a girl aged ten years is more convincing. Guild, Kindell and Gibson[15] collected from the literature records of twenty probable cases of malignant hypertension in children, and added two of their own. In twelve the cause of death was known. In four of these it was renal failure. Nye found a large number of cases of chronic nephritis in young people in Queensland, and traced the source to lead poisoning in many of them. Fishberg[11] has stated, and it is agreed upon by many writers, that lead is preeminent among the exogenous poisons which cause chronic hypertension in man. It is probable then that in many of Nye's cases lead was responsible for hypertension and eventually for chronic nephritis, and Duhig's pathological reports confirmed this. Calvin and Rosenblum[6] go so far as to say that in about 10% of cases of primary hypertension the sclerosis of the renal arteries leads to renal insufficiency and uræmia.

Even in these cases it is difficult to exclude a congenital abnormality of the kidneys as the underlying ætiological factor. Schwartz[39] reported three cases of malignant hypertensive nephritis, only one of which came to autopsy, and a congenital aplasia (kidney practically absent) on one side was found, with subacute nephritis in the kidney on the other.

Syphilis.

Earlier writers, notably Sawyer[40] and Guthrie,[18] favoured a syphilitic origin for chronic nephritis. This was, however, before the introduction of the Wassermann test. Present-day authors have been unable to establish a connexion between the two.

Recurring Pyelonephritis.

It is extremely doubtful whether during childhood chronic recurring pyelonephritis affecting in a healthy kidney can eventually cause such a degree of renal fibrosis as to lead to chronic renal insufficiency. In Spence's[44] classification "chronic ascending pyelonephritis" forms one group of cases. His cases did not include one of this nature, and he cites those of Young, Frontz and Baldwin,[47] following valvular obstruction of the posterior part of the urethra of male children. Spence goes on to say that "it should be asked if there is not some chronic obstruction or deformity of the urinary tract which is the cause of the infection, for in children, as in adults, imperfect urinary drainage plays an important role in this". The answer would appear to be in the affirmative.

Pepper and Lucke[35] emphasized the view that when one kidney is congenitally absent or hyperplasia exists the apparently normal kidney is especially prone to chronic inflammatory processes; and it is well known that congenitally defective kidneys are more vulnerable to infection than the normal kidney. It would probably be preferable to place this group among the congenital abnormalities associated with chronic nephritis.

Surgical Conditions of the Kidneys.

It is conceivable that surgical conditions, such as calculous nephritis, obstruction by tumour of the ureter with pyonephrosis *et cetera*, may ultimately lead to such fibrosis of the renal tissues as to give a clinical picture of chronic nephritis; but in childhood these conditions are rare, and usually terminate in death from some other complication before such a stage is reached.

Summary of Ætiological Considerations.

We may say, therefore, that the pathologico-clinical end-result may be reached in several different ways:

1. In association with congenital abnormalities of the urinary tract, for example, hypoplasia, hydronephrosis, hydroureter, ureteral stenosis, urethral valve, polycystic kidneys, achalasia of sphincters, especially when these are complicated by repeated infection of the kidneys, the so-called "chronic pyelitis".
2. As the terminal stage of an acute or subacute nephritis.
3. From renal destruction in malignant hypertension.

The congenital group is a fascinating one. Congenital malformations of the kidneys may give no evidence of their presence during life and may be discovered only *post mortem*. These are of academic interest only and do not concern the present discussion.

The second group of congenital lesions is associated with sudden death in infancy after a vague illness or after operation. Again, one is surprised to find the kidney lesion at *post mortem* examination; but these differ from the former in that it is the renal failure which has been responsible for the patient's demise.

It is well known that infection is apt to be superimposed on these congenital abnormalities, and these cases are notoriously difficult to clear up. With recurrent attacks the function of the kidney becomes more and more impaired, until death ensues from exhaustion or renal failure. Such cases constitute the third group.

Finally, the fourth group of congenital abnormalities may cause the signs and symptoms of chronic renal failure from the start and terminate in uræmia. Cases of renal rickets and renal dwarfism are generally of this nature; and life, in spite of the rising blood urea content, is strangely prolonged, allowing such skeletal changes to ensue.

All except the first group would fall into a clinico-pathological classification of chronic nephritis.

The cases satisfying these postulates occurring at the Children's Hospital, Melbourne, from 1932 to 1937 will be reviewed and placed in their respective groupings. Special points of interest arising out of these cases will be discussed.

Case Reports.

Congenital Abnormalities Giving Rise to Chronic Nephritis.

Sudden Death in Infancy.

Case I.—D.W., a female infant, aged nine months, died after a minor operation for tuberculous glands in the neck. Before the operation there had been no suggestion of renal dysfunction. At *post mortem* examination the heart was small and not hypertrophied; the kidneys were definitely granular, with multiple small cysts projecting from the surface. The general appearance was not that of a congenital polycystic kidney, the cysts being very small, the cortex narrow, and the substance of the kidney tough and granular, with poor definition between cortex and medulla.

Case II.—S.S., a female baby, aged eleven weeks, had difficulty with its feeding since birth, would not take the breast, vomited cow's milk mixture, and failed to put on any weight. As the vomiting was projectile, pyloric stenosis was thought a possible cause, but was excluded by the absence of visible peristalsis and the absence of a tumour. The baby died from inanition, and at *post mortem* examination the only finding was two very small atrophic kidneys with some dilatation of one ureter and pelvis.

Case III.—L.A., a baby boy, aged two weeks, was the result of a second pregnancy after a normal confinement. The accoucheur noticed that after birth the baby cried feebly and that the whole of the abdominal contents flopped over to the side most dependent. There was also bilateral talipes. The heart and lungs were normal. The abdomen was lax and wrinkled. The abdominal wall was very thin, consisting apparently only of skin and subcutaneous tissue. The stomach and intestine could be seen contracting beneath the external covering. The baby soon came to *post mortem* examination, when the complete absence of abdominal muscles was verified. There was a large diverticulum of the bladder, which could be felt clinically. The ureters were extremely dilated and tortuous, approximating the small intestine in calibre. The right kidney was rudimentary, whilst that on the left side was enlarged to about twice the normal size. The pelvis of the rudimentary kidney was very much dilated, as were the pelvis and calyces of the enlarged kidney on the left side. No obstruction or stenosis could be found anywhere in the renal tract, and a probe was easily passed up the urethra.

Case IV.—R.C., a male infant, aged six months, was admitted to hospital with a history of difficulty in feeding, vomiting and some diarrhœa. On physical examination nothing abnormal was determined except that the infant was undersized. The urine contained numerous bacilli and a few pus cells. Apart from this there was no reason to suspect the kidneys; but the infant died suddenly, and at *post mortem* examination the right kidney was found to be very atrophic, about the size of a walnut, with dilated pelvis and ureter. There was no sign of stenosis at the pelvi-ureteral or uretero-vesical junction. The left kidney was larger than normal, with accentuation of fœtal lobulation.

These cases constitute a very interesting and instructive group. They afford very good evidence of the congenital origin of these abnormalities. King and Sheldon[23] described a similar case in which death occurred at the early age of three weeks, and at *post mortem* examination bilateral hydronephrosis and gross destruction of the kidneys, due to valvular obstruction of the urethra, were found.

Such cases demonstrate too the extreme difficulty in arriving at the correct diagnosis during infancy. A "feeding difficulty" or "dietetic dyspepsia" or "arthrepsia" is the diagnosis most frequently pinned onto such cases. Changes of food and modification of the elements, however, fail to ameliorate the vomiting and loss of weight. They are indeed a very complex and worrying problem to the attending physician.

It is obvious that the ultimate fate of the patient depends upon the degree of involvement of the kidneys. The fibrosis may be of such an extent as to allow life to be carried on at a low ebb; but as soon as some strain is thrown upon such kidneys they fail to function adequately enough to prolong life.

Congenital urinary abnormalities are not an infrequent *post mortem* finding. Sheldon[43] found them in 2% of autopsies. All cases, however, cannot be classed as chronic nephritis. It is when renal failure appears clinically or no other cause is found at autopsy to account for death that they may be given this title.

The association of hydronephrosis, hydroureters and dilatation of the bladder with congenital absence of the muscles of the abdominal wall, as occurred in Case III, is a well-known abnormality.

There may be many authorities who object to the inclusion of these cases under the title of chronic nephritis, but as death can be explained only by the renal failure; and since on histological examination the kidneys of chronic insidious nephritis and congenital atrophy have many points in common, such a grouping appears justifiable. In one of Ellis and Evans's[8] cases the baby came to *post mortem* examination at three weeks. In all their cases they found the interpretation of the histological findings very difficult, the kidneys showing "in addition to the signs of hydronephrosis with resulting compressive atrophy, the appearance which we associate with a nephritis".

Congenital Abnormalities with Infection.

Case V.—G.D., a boy, aged seven and a half years, gave a past history of bronchopneumonia nine months previously and of nocturnal enuresis since birth. For the five days prior to his admission to hospital he had a "heavy cold", and for several weeks some increased frequency of micturition by day. He was a well-nourished boy, the tongue was furred, the heart appeared to be normal, the lungs showed a recurrence of the bronchopneumonia, but nothing abnormal was detected in the abdomen. The urine contained albumin, organisms (streptococci), pus cells and a few red blood cells. He gradually recovered from his lung condition, but his renal disability persisted.

The urea concentration test showed an output of 0·55% urea in urine after one hour, 0·70% after two hours, and 0·81% after three hours. His blood urea content was 23 milligrammes per hundred cubic centimetres. The Wassermann test applied to his blood elicited no reaction. A plain skiagram of his renal tract showed no obvious abnormality. Cystoscopy revealed cystitis. He developed a brawny thickening in the right groin, which was very tender to palpation, and exploration showed it to be due to pyoureter with surrounding reactionary œdema. This was present on the left side too. The ureters were incised and drained, but death supervened. At autopsy the ureters were both grossly dilated and tortuous. Bilateral pyonephrosis was present. No constriction of the ureters was demonstrable. The kidneys were extremely lobulated, the left being very small indeed. On section there was no visible differentiation between cortex and medulla. The bladder showed acute cystitis. The *post mortem* diagnosis was congenital pyonephrosis and pyoureters, chronic nephritis, pyelonephritis and cystitis.

Case VI.—C.H., a girl, aged eight years, was seen first in April, 1935. She had suffered from enuresis since birth. Her urine contained albumin and microscopically a large number of pus cells and bacilli were seen. Excretion pyelography revealed extremely poor excretion of the dye on the right side. Cystoscopy and retrograde pyelography showed well-marked evidence of cystitis, especially around the right ureteric orifice, and a definitely abnormal shadow of the right renal pelvis. The urine from this side contained pus cells, but there was no growth of organisms on attempted culture. The urine from the left pelvis contained red cells only (instrumental). At this stage the diagnosis of a congenital abnormality of the right kidney with superimposed infection was made. The child was readmitted to hospital a year later. There was still a moderate amount of albumin in the urine. Microscopically pus cells, numerous organisms and a few red blood cells were discernible. The concentration of urea in the urine was 1·45% after one hour, 2·05% after two hours, and 2·10% after three hours. The blood urea content was 38 milligrammes per hundred cubic centimetres. It was decided to excise the right kidney. The specimen was an atrophic kidney with double pelves and ureters and some degree of hydronephrosis and hydroureters. The patient succumbed soon after the operation. The remaining kidney gave the appearance of acute hæmorrhagic nephritis. There was a large vesical diverticulum and severe cystitis.

Case VII.—B.M., a male, aged ten years, began his illness with an acute pyelitis with rigors, the urine containing albumin, pus cells and bacilli. This responded poorly to alkalinization and later to acidification. He remained for some months in indifferent health and was subject to severe attacks of vomiting, dyspnœa and drowsiness, which were thought to be uræmic. Indeed the blood urea content was found to be increased, being 114 milligrammes per hundred cubic centimetres on occasions; and although this level dropped in the intervals to normal, the urea concentration tests showed impaired renal function—0·85% after one hour, 0·80% after two hours, and 0·90% after three hours. Attempt at cystoscopy was unsuccessful and excretion pyelography was considered to be dangerous. It was felt that the condition was one of chronic nephritis associated with some congenital abnormality upon which "pyelitis" was superimposed.

It is well recognized that when pyuria is chronic, or when so-called "pyelitis" has persisted more than three months, there is often some underlying anatomical abnormality in the kidneys, though, of course, other conditions, such as tuberculosis, calculus formation, cord lesions, neoplasms of the kidney and bladder, vertebral or appendiceal abscess and disease of the pelvic tissues, may possibly be the cause. But a congenital defect is the most frequently met with. The introduction of mandelic acid therapy by Rosenheim[37] has served to accentuate this opinion in those cases which are resistant to treatment.

It may be questionable whether pyelonephritis occurring in a previously healthy kidney could eventually lead to such fibrotic changes as to produce the signs and symptoms of chronic renal failure; but it is certain that such an occurrence in a kidney already sclerosed and scarred would need little associated inflammation to impair its function gravely.

Some physicians would include Case VII under this heading, but the frequency with which some congenital lesion can be demonstrated either during life by pyelography or *post mortem* militates too strongly against this view.

Again, there will be many purists who will not agree with a classification of such cases under a heading of chronic nephritis; but surely it is justifiable in those cases that during life present the clinical features of chronic renal failure, and also in those cases in which *post mortem* examination shows that the fibrosis was an apparently important factor in causing death. Numerous cases of renal atrophy or fibrosis with superimposed infection have been excluded from these case reports because they did not satisfy these postulates.

Congenital Abnormalities with Chronic Renal Failure.

Case VIII.[1]—L.C., a male child, aged one year and six months, presented the classical features of rickets, notably *genu valgum*, bowing of forearms, beading of the ribs and a widely open fontanelle. At the age of four months the deformity of the chest suggested rickets, but there was little or no response to the usual antirhachitic treatment. Over a period of twelve months there was scarcely any gain in weight. At eighteen months the patient was noticeably dwarfed. A renal origin was suspected. The urine contained no albumin (one examination only), but the blood urea content was 155 milligrammes per hundred cubic centimetres, the blood phosphorus was 6·2 milligrammes per hundred cubic centimetres, and the blood calcium was 7·9 milligrammes. The blood did not react to the Wassermann test, and the findings on Röntgen ray examination of his bones were consistent with a diagnosis of renal rickets. He came to autopsy twelve days after his admission to hospital, and examination of the urinary tract showed that both ureters were moderately dilated in their whole course, except near the bladder, where they were patent and of normal size. Pronounced internal and external hydronephrosis and thinning of the renal substance in both kideys were present. The kidneys were small and pale, with an irregular surface; the capsule was slightly adherent. The bladder was distended. The posterior part of the urethra showed a valve-like obstruction at the distal end of the verumontanum, with dilatation of the urethra proximal to the obstruction.

Case IX.—J.L., a girl, aged eleven years, was admitted to hospital in a comatose condition after a vague illness lasting one month. The main symptoms were vomiting, great weakness, hæmaturia and diarrhœa with blood and slime in the motions. She had been stuporose for the last twenty-four hours. On examination she was pale, her eyes were fixed and staring, there was twitching of the limbs, the respirations were deep and slow, the breath was uriniferous, the tongue was dry and brown, and the pupils were dilated and sluggish in their reaction to light. The heart was enormously enlarged; there was gross papilloedema, and retinal hæmorrhages and exudate were visible in both fundi. The systolic blood pressure was 190 millimetres of mercury and the diastolic pressure 100

[1] This case has been described more fully, especially from the pathological viewpoint, by Reginald Webster in his series of reports published in this journal.

millimetres. She died without recovering consciousness, and at autopsy her right kidney was "very atrophic and so small that it could be placed in the palm of the hand". The capsule was adherent everywhere. On section it was difficult to distinguish cortex from medulla in the general fibrosis. The left kidney was, on the contrary, very large, but was also extremely fibrotic, with adherent capsule. It too showed poor definition between cortex and medulla. There was no dilatation of the renal pelvis on either side, and no malformation of the ureters. In the heart great left ventricular hypertrophy was present. The colon contained much blood, the mucous membrane being extremely infected with sloughing at places.

Case X.—C.H., a female child, aged eleven years, was first seen in a convulsion. Her parents stated that she had enjoyed good health till one day prior to her admission to hospital, when she complained of headache, was feverish and vomited a good deal until twitchings set in and she became difficult to arouse. On examination her systolic blood pressure was 240 millimetres of mercury and her diastolic pressure 160 millimetres. Nothing abnormal was detected in the heart or lungs, but examination was a very difficult matter. There was a doubtful strabismus, the knee jerks were hyperactive, the superficial abdominal reflexes were not elicited, and Babinski's sign was present on the right side. The fundi showed bilateral papilloedema, hæmorrhages and exudate. The urine contained macroscopic blood and was loaded with albumin. Microscopically pus cells and bacilli were also discovered, but no casts were seen. Her condition improved enough for her to be allowed home, but she had to be readmitted to hospital a month later in uræmia. The systolic blood pressure now was 226 millimetres of mercury, and the diastolic pressure 175 millimetres. The apex beat of the heart was well outside the nipple line, and the second sound was accentuated at the base. The findings on examination of the urine were similar to those described above. The blood urea content was 100 milligrammes per hundred cubic centimetres. Her condition again improved and she was allowed to go home, but was readmitted to hospital two months later and died in uræmia, the principal symptom being intense dyspnœa. She came to autopsy. Her heart, spleen and liver were larger than normal. The lungs showed bilateral basal congestion and were both very œdematous. There was bilateral effusion into the pleural sacs. The left kidney was represented by a small hydronephrotic remnant in which there was practically no renal tissue. No obstruction to the ureter could be demonstrated along its length. There was a mild degree of congenital hydronephrosis on the right side. The capsule of this kidney was slightly adherent, but the surface was not granular. There was poor definition between cortex and medulla on section. The pathologist concluded that chronic nephritis had supervened on a mild degree of congenital hydronephrosis and an acute infection brought about a rapid termination.

Case XI.—J.E., a little girl, aged four years and six months, was admitted to hospital with a history of puffiness under the eyes and the passage of small amounts of darkish urine for four days. Examination revealed a slightly infected throat, but no œdema could be demonstrated at this time. The urine was blood stained and loaded with albumin. No pus cells were seen under the microscope. A sudden demise precluded further investigation, and *post mortem* examination revealed a very atrophic left kidney, measuring not more than 3·75 centimetres from pole to pole. The renal calyces and pelvis of the kidney remnant were dilated, but the ureter not noticeably so. No obstruction to the ureter could be demonstrated. The right kidney showed compensatory hypertrophy. Chronic nephritic changes in this organ were indicated by an adherent capsule and very narrow cortex, where cortex tissue could be distinguished from the medulla. The whole organ was tough and friable. The renal pelvis was also dilated on this side, but again there seemed to be no dilatation or stenosis of the ureter. The left ventricle of the heart was hypertrophied. The pathologist diagnosed abiotrophy of the left kidney, an acute exacerbation on a chronic nephritis.

Case XII.—D.E., a female child, aged thirteen years, without previous illness, gave a history of vomiting, headache, some frequency of micturition and blurred vision during the previous three or four months. On examination she was pale and it was thought there was some degree of exophthalmos. The apex beat of the heart was 2·5 centimetres outside the left nipple line, and a systolic murmur was audible, maximum at the apex. The systolic blood pressure was 185 millimetres of mercury and the diastolic pressure 140 millimetres. The urine showed no blood macroscopically and gave a faint deposit of albumin on boiling. Microscopically a few red blood corpuscles, pus cells and granular casts were visible. The ocular fundi revealed bilateral papilloedema, hæmorrhage and exudate. The blood urea content was 31 milligrammes per hundred cubic centimetres; the urea concentration test gave 1·00% urea after one hour, 0·90% after two hours, and 1·10% after the third hour. The blood chloride content was 455 milligrammes per hundred cubic centimetres (normal 450-500 milligrammes). The plasma protein was 9·0 grammes per hundred cubic centimetres. Excretion pyelography revealed gross hydronephrosis on the left side and no excretion of the dye by the right kidney. There was no dye visible in the bladder up to fifteen minutes; this suggested deficient renal function and nonpatency of the left ureter. Cystoscopic examination revealed a patulous left ureteric orifice. The catheter passed up the right ureter for about three centimetres, but could be passed no further. A catheter passed a similar distance up the left ureter, and after it had encountered some obstruction was passed on. Retrograde pyelography verified the finding of left hydronephrosis and demonstrated left hydroureter in addition. The right pelvis was not very well filled, but the outline was definitely abnormal.

Investigation three months later showed a blood urea content of 75 milligrammes per hundred cubic centimetres; the blood chlorides were 350 milligrammes, and plasma protein was seven grammes per hundred cubic centimetres. The diagnosis was congenital malformation of the renal tract with superimposed chronic nephritis.

Case XIII.—N.L., a girl, aged twelve years, had a past history of recurrent attacks of "pyelitis" four years prior to her present illness. She was admitted to hospital with a history of having had a fit the night before. Vomiting, fever and delirium were conspicuous features of the illness one day before this. Headache set in and her vision became blurred on the day of her admission. She was stuporose on examination. Her apex beat was 2·5 centimetres outside the nipple line in the fifth left intercostal space. The systolic blood pressure was 170 millimetres of mercury. The urine was loaded with albumin, and microscopically a few pus cells, red blood corpuscles and abundant hyaline and granular casts were demonstrable. The blood urea was 95 milligrammes per hundred cubic centimetres. The report on her fundi was "albuminuric retinitis". Her urea concentration was 0·90% after one hour, 1·35% after two hours, and 1·25% after three hours. Cystoscopic examination revealed a rather œdematous right ureteric orifice, but the orifice on the left side was normal. The ureters were easily catheterized. Urine from the right pelvis contained pus and organisms, which on culture proved to be *Staphylococcus albus*. Urine from the left pelvis was clear and gave no growth to incubation. Retrograde pyelography showed a moderate degree of hydronephrosis on the right side, with some "cupping" of the calyces. The left pelvis appeared flat and deformed, with no definition of the calyces.

The patient was lost sight of, but at this stage it was thought that she had congenitally malformed kidneys and that, with recurring infection taking place, chronic nephritis and chronic renal failure were imminent.

Many cases of renal rickets similar to Case VIII have been described. In some of these there has been no demonstrable obstruction to account for the hydronephrosis, hydroureters and dilated bladder. Ellis and Evans's[8] group of cases was notable in this respect. In others, as in Case VIII, an anatomical

obstruction to the flow of urine was revealed at autopsy, a congenital urethral valve being the usual offender. For this reason Poynton and Sheldon[48] stress the importance of the removal of the urethra in continuity with the bladder in *post mortem* examination in all cases in which urinary tract dilatation is present. Ellis and Evans were careful to carry out this procedure. In their cases they suggest a disorder of the neuro-muscular mechanism controlling the urethro-vesical sphincter.

In Cases X, XI, XII and XIII hydronephrosis was proved to be present in some degree, and in all this condition was accompanied by rise in the blood pressure. The difficulty arises in classifying similar cases in which, however, no actual congenital abnormality is demonstrable, but in which a small fibrotic kidney is found. This may be due to congenital abiotrophy or to hypertensive changes. When the atrophy is extreme so as to approach the well-known congenital absence of a kidney, as in Case IX, there should be no doubt; but borderline cases will undoubtedly arise which will cause no end of confusion and uncertainty.

A superimposed acute or subacute infection may complicate the clinical picture, as shown by the hæmaturia in Case X, and the œdema in Case XI; but this should not mislead us.

The frequency with which the condition in this group of cases is mistaken for some cerebral catastrophe, such as cerebral tumour or encephalitis, only serves to emphasize the necessity for more frequent estimation of the blood pressure and more careful examination of the urine during childhood.

It will be observed that the majority of cases of chronic nephritis—thirteen out of seventeen—in these reports are associated with some congenital lesion; and this has been the conclusion reached by other observers.

The classification given demonstrates the form in which congenital lesions of the kidney may present themselves to the physician or possibly to the surgeon. It is hoped that by this means the condition may be more frequently recognized and the medical attendant may be in a position to give a guarded prognosis at the earliest possible time.

Chronic Nephritis as the Terminal Stage of an Acute or Subacute Nephritis.

Case XIV.—C.D., a boy, aged seven years and eight months, was admitted to hospital with an acute hæmorrhagic nephritis without œdema and with petechiæ on various parts of the body. Both the findings were apparently caused by septic tonsils. He recovered after spending three weeks in hospital and was discharged with a view to tonsillectomy later. Three days after leaving hospital he developed a severe hæmaturia, with œdema of the face and ankles, and at this time ascites was demonstrable. The hæmaturia became insignificant, but the anasarca and ascites increased, until he presented the picture of "subacute nephritis" or the nephrotic syndrome. This subsided after four or five months and he attended the out-patient department in indifferent health. It was noticed that growth was at a standstill. X ray examination of the epiphyses showed no evidence of renal rickets. Two and a half years after his original illness it was apparent that chronic nephritis was setting in. He suffered from frequency of micturition, vomiting, headache and eventually drowsiness. He was grossly underweight and undersized—1·5 kilograms (three stone three pounds), compared with normal 2·2 kilograms (four stone twelve pounds). The blood urea content rose to fifty-one milligrammes per hundred cubic centimetres, and later reached over 165 milligrammes on three occasions. His urea concentration was 0·7% after one hour, 0·9% after two hours, and 1·0% after three hours. His systolic blood pressure rose to 148 millimetres of mercury and the diastolic pressure to 98 millimetres. The apex beat was displaced outwards, a soft systolic murmur was audible, and the second sound became accentuated at the base. The urine contained albumin varying in amount from a trace to a fairly heavy deposit. Microscopically occasional red blood cells and hyaline casts were seen. A small retinal hæmorrhage appeared on the right fundus below the optic disk. During this time too the clinical signs of tetany became manifest, the patient's hands being held in the accoucheur position and Trousseau's sign being positive. The serum calcium content was 4·6 milligrammes per hundred cubic centimetres, and the plasma phosphorus 8·3 milligrammes. The blood showed severe anæmia, the red blood corpuscles numbering 2,350,000 cells per cubic millimetre and the hæmoglobin value being 33%. The plasma protein content was 4·2 grammes per hundred cubic centimetres. The alkali reserve was 43%—a mild degree of acidosis. X ray examination of the epiphyses at this stage showed no evidence of renal rickets.

The patient died in uræmia complicated by the onset of pericarditis three years and seven months after the onset of the illness. At the *post mortem* examination there was pulmonary œdema with bilateral hydrothorax, the amount of fluid present not being excessive. The heart showed hypertrophy of the left ventricle and pericarditis. The kidneys had thickened capsule, which stripped with occasional tearing of the cortex. The cortex was finely granular. There was no malformation of the urinary tract.

Case XV.—D.C., a girl, was first seen at the age of six years and eleven months. Her previous illness was tonsillitis, from which she had suffered two years previously. As far as could be ascertained this was not complicated by nephritis. There was œdema of the legs and abdominal wall, and some degree of ascites and hydrothorax was present. The urine contained a large amount of albumin, and microscopically numerous granular casts and a few leucocytes were seen. The blood urea content was 39 milligrammes per hundred cubic centimetres, and the urea concentration test gave 2·2% after the first hour, 1·63% after the second, and 2·64% after the third.

The anasarca, ascites and hydrothorax responded to a certain extent to diuretics and repeated paracentesis, but recurred on several occasions. Acute exacerbations occurred, when numerous blood cells appeared in the urine, the blood urea content rose above 100 milligrammes per hundred cubic centimetres, and the urea concentration test gave percentages below 2·0.

Ultimately frequency and nocturnal micturition became a troublesome feature, languor and drowsiness became prominent and the patient was admitted to hospital and died in convulsions three years and eight months after her original illness. On examination just before death she was sallow and comatose. There was no œdema. The tongue was dry and brown. Lumbar puncture yielded a clear cerebro-spinal fluid under increased pressure. The urea in the cerebro-spinal fluid was 150 milligrammes per hundred cubic centimetres. Time did not permit of further investigation and a *post mortem* examination was refused.

Case XIV is one of those interesting cases which was studied as it passed through all the stages of glomerulonephritis during a period extending over three and a half years. Five to six years is perhaps the usual length of span of such cases, but the limits vary widely between six months and forty years. Mann's[27] patient was under observation for twenty-eight years.

It is cases such as these that have been chiefly instrumental in changing our conception of nephritis from that of a confusing variety of distinct diseases with water-tight compartments to one of a single continuous process passing through stages recognizable clinically and pathologically. What an instructive picture we should obtain if by some magic process we were able to take a continuous film of the histological changes taking place in the kidneys of the patient described and to speed it up from three and a half years to three and a half hours! Actually we do the next best thing and take a series of "stills" from different cases in the *post mortem* room and laboratory, and when we piece them together, although many pieces may be missing, the picture, like a jig-saw puzzle, appears before our eyes.

Perhaps the following summarizes the possible sequences that should be kept in mind in such cases: Acute nephritis (first stage) may be followed by recovery, by subacute nephritis (second stage) or by death, or there may be subsequent attacks of acute nephritis. Subacute nephritis in its turn may be followed by recovery, by chronic nephritis (third stage) or by death, or there may be subsequent attacks of subacute nephritis.

The first stage may drift insidiously into the third stage without the appearance of obvious signs of the second stage. The first stage may be overlooked or absent, and the patient may first appear with subacute nephritis (second stage), and the sequences may then follow as described above. Both the first and second stages may have been absent and the patient may appear with the signs and symptoms of the third stage or chronic nephritis. Finally there may be exacerbations of the signs and symptoms of acute nephritis during the second and third stages.

The onset of the disease in Case XIV is also worthy of attention. Apparently toxins elaborated from the septic tonsils so damaged the endothelium of the skin, bowel and renal capillaries as to produce purpura, bowel hæmorrhages and hæmaturia. It was not until the child had been allowed home and returned with severe hæmaturia that it was realized that the brunt of the attack had fallen on the sensitive renal capillaries and the seriousness of the case became apparent. It is possible that had it been opportune to eradicate the septic focus the disease might have been arrested. Playfair[36] records two interesting cases of thrombopenic purpura associated with tonsillar infection in which recovery was complete after removal of the focus of infection. In these, however, the renal capillaries were not attacked.

The presence of dwarfism in these cases of renal insufficiency has been commented on by numerous writers since Lucas[25] in 1883 published his paper "A Form of Late Rickets Associated with Albuminuria". This patient was noticeably dwarfed, but the absence of "rhachitic" changes at the epiphyses, in spite of the low serum calcium content, and the great upset in the calcium-phosphorus ratio are not easy to understand. Probably the explanation lies in the fact that growth has been at a standstill; and indeed Sir Frederick Still and Leonard Parsons[33] have frequently pointed out that in cœliac disease rickets will not appear until growth begins to take place during a remission or as improvement sets in.

The exhibition of tetany in this case is of more than passing interest. Chronic nephritis is one of the rarest causes of tetany and is, of course, explained by the great reduction in serum calcium. Actually it is the amount of "free" or ionized calcium which governs the appearance of tetany. Lathrop[24] has recorded a case in which the total calcium of the serum was very low, but in which tetany did not occur because the "free" calcium as shown by estimation was normal.

Ross and Scriven[38] have shown that the occurrence of acidosis leads to the mobilization of calcium ions from the calcium reservoirs. De Wesselow[7] has shown that the acidosis in nephritis favours the ionization of the diminished amount of calcium present. It would therefore appear that the acidosis plays a part in the infrequent occurrence of tetany in cases of chronic nephritis and aggravates the more commonly seen rickets. In the case under discussion the acidosis apparently did not reach a degree sufficient to raise the "free" calcium in the serum to avert the appearance of tetany.

In Case XV we are without autopsy proof that the disease in the kidneys had reached the third stage, but the clinical findings, especially the absence of œdema, leaves little doubt that this has actually occurred.

Renal Destruction in Malignant Hypertension.

Case XVI.—E.S. was first seen as a little girl, aged five years, suffering from abdominal colic, secondary anæmia with basophilic stippling and double foot-drop, apparently due to lead poisoning. Von Pirquet's tuberculin test and the Wassermann test of the blood elicited no reaction, and X ray examination of the long bones revealed no abnormality at this time. Unfortunately the blood pressure was not recorded at this stage, but the examination of the urine on three occasions showed it to be clear of albumin, and the specific gravity varied between 1012 and 1020.

She was next seen when she was suffering from left basal pneumonia at the age of nine years. A soft blowing systolic murmur was recorded over the apex of the heart at this stage. Again the blood pressure was not recorded nor the urine examined.

Four years later, at the age of thirteen, she was found to have a systolic blood pressure of 170 millimetres of mercury and a diastolic pressure of 128 millimetres. The apex beat was in the nipple line in the fifth intercostal space. There was an apical systolic murmur and the basal second sound was accentuated. Examination of the urine revealed slight albuminuria. The blood urea content was 63 milligrammes per hundred cubic centimetres. The urea concentration test gave the figure 1·75% after the third hour. The report on the fundi was "arteriosclerotic". Fifteen months later, headache, vomiting, twitchings and polyuria were the principal complaints. The patient was pale, the systolic blood pressure was 198 millimetres of mercury, and the diastolic pressure 160 millimetres. The urine contained a moderate amount of albumin, and microscopically a few granular casts and an occasional red

blood corpuscle were seen. The specific gravity was fixed in the region of 1010. The report on the fundi was "albuminuric retinitis". The blood urea content rose to 164 milligrammes per hundred cubic centimetres, and the child ultimately died in uræmia.

At autopsy the left ventricle was found to be grossly hypertrophied. The kidneys were a little contracted in size, though not conspicuously so. The capsule stripped with great difficulty and resulted here and there in tearing of the kidney substance. On section it was extremely difficult to define cortex from medulla. The surface was finely granular and patches of diffuse fibrosis resulted in a pallid general effect.

CASE XVII.—M.H., a girl, aged ten years, gave a history of drowsiness, headaches and occasional vomiting with some swelling of the eyelids during the previous three weeks. Over the previous twelve months she had suffered from two or three attacks similar to this. Examination revealed a pale girl with puffy eyes and numerous skin petechiæ. The heart was hypertrophied, the spleen was just palpable, the systolic blood pressure was 204 millimetres of mercury and the diastolic pressure 160 millimetres. Examination of the ocular fundi revealed bilateral papilloedema, hæmorrhages and much exudate. The urine was blood stained and loaded with albumin. The blood urea content was 101 milligrammes per hundred cubic centimetres. The blood did not react to the Wassermann test. She succumbed during this illness, and at autopsy the size of the heart was verified, the increase being due mainly to left ventricular hypertrophy. The kidneys were larger than normal. On the surface both showed depressed patches over which the capsule was adherent. The findings were consistent with a subacute exacerbation on a hypertensive nephritis.

It will remain a matter of conjecture whether in these cases the condition is really hypertensive nephritis or congenital abiotrophy of the kidneys which has been followed by increase in blood pressure and renal failure. The histological findings should be of some help in this respect, but unfortunately are not reliable. An adequate history would also be of some assistance, but this is rarely forthcoming. In Case XVI we have a history of lead poisoning, which is a well known cause of "nephrosclerosis", and its inclusion under this heading is justifiable on these grounds.

The same difficulty has been encountered by others. Schwarz[39] reported three cases of malignant hypertensive nephritis, in only one of which an autopsy was allowed. In this case a congenital aplasia was found in one kidney and a subacute nephritis in the other. He did not think, however, that in all cases a congenital abnormality was the underlying causative factor. Some he thought had an "hereditary predisposition".

Guild, Kindell and Gibson[15] collected from the literature records of twenty possible cases of malignant hypertension in children, and added two of their own. Congenital hypoplasia of the kidneys was present in two cases only.

Acute or subacute infection superimposed on these already poorly functioning kidneys is often, as in Case XVII, the immediate cause of death.

SUMMARY.

The literature on chronic nephritis in childhood is reviewed, special attention being paid to its ætiology.

A classification on an ætiological basis is given, and cases occurring at the Children's Hospital, Melbourne, during a five-year period, are grouped under their appropriate headings.

Special attention is paid to the manner in which patients so affected may present themselves to the physician and surgeon. It is hoped that by this means the condition may be more frequently recognized at the earliest possible moment and a guarded prognosis given.

To this end a plea is made for more frequent urinary examination and blood pressure estimations in children.

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Reports of Cases.

DISORDER OF MATERNAL CARBOHYDRATE METABOLISM COMPLICATING PREGNANCY.

By A. M. Davidson, M.B., Ch.M. (Sydney),
Enmore, New South Wales.

The patient was aged twenty-seven years and had been married for four years. Inquiry into her family and personal history revealed nothing of note. She had had no miscarriages. Her last menstrual period had occurred on June 12, 1938. Her history as regards child-bearing was that she had been confined twice in the country, and on each occasion had been delivered by forceps of an abnormally large child. The infants had weighed 4·8 and 5·8 kilograms (ten and a half and twelve pounds) respectively; both had been still-born from disproportion and the resulting birth injuries. The patient stated that on each occasion the rate of increase in size of her abdomen during the last two months of pregnancy had, in the opinion of her medical adviser, been very much greater than normal, and that premature induction of labour had been attempted, but had failed. I was unable to obtain any evidence that sugar, acetoacetic acid or acetone had been found in her urine in either pregnancy.

Her height was 147·5 centimetres (four feet eleven inches) and her normal weight 50·8 kilograms (eight stone). Her external pelvic measurements were as follows: intercristal, 22 centimetres; interspinal, 26 centimetres; bitrochanteric, 30 centimetres; external conjugate, 19 centimetres.

In view of the patient's history of still-born babies, apparently through disproportion, I had advised her after her second confinement to come to the city for her third and to have a Cæsarean section. She came to the city at the thirty-second week. Up to that time her pregnancy had apparently been normal, except that she had felt the heat wave very much at her home in the central west of New South Wales; she had collapsed one very hot day and had needed medical attention. She stated that since then she had consumed large quantities of ice cream and sweet drinks.

On February 3, 1939 (thirty-third week), a specimen of urine was examined. The specific gravity was 1054, and the urine was found to contain 10% of glucose (according to the pathologist's report) beside acetoacetic acid and acetone. I immediately sent her to hospital and administered 20 grammes of glucose and 10 units of insulin. The next morning the urine was sugar-free; but ketone bodies were present. She was then ordered a diet of about 1,100 Calories, with 100 grammes of carbohydrate and five units of insulin before each meal. The urine then contained a trace of sugar; but acetoacetic acid and acetone were present in disquieting amounts. As her fasting blood sugar content had been as low as 88 milligrammes per 100 cubic centimetres, indicating a low renal threshold, caution was exercised in increasing the insulin dosage; her diet was increased to 1,300 Calories, with additional glucose after each dose of insulin. The ketone bodies still persisted after her urine was sugar-free.

Her diet was further increased to a normal full diet with no restrictions; and when the dosage of insulin was increased to 40 units *per diem* in four doses her urine became free of sugar and ketone bodies. In view of the unfortunate results of her previous deliveries, and the fact that the fœtus was now quite large and vigorous (as determined by palpation and auscultation of the fœtal heart), it was decided to deliver her by Cæsarean section rather than to run the risk of further rapid increase in size of the fœtus and of the necessity for administering still larger doses of insulin. Moreover, her psychological state was unsatisfactory, as she was very anxious about her condition and very depressed as to the outcome. She required potassium bromide to enable her to sleep.

On February 11, 1939, the operation was performed by the classical method, 20 grammes of glucose and 10 units of insulin having been administered two hours before. A dose of 0·4 milligramme (1/150 grain) of atropine sulphate was given half an hour before. The child, a female, weighed four kilograms (eight pounds fourteen and a half ounces). It was very fat, the general appearance being that of a full term infant; the only indications of prematurity were the presence of lanugo and the development of the nails. Unfortunately it survived only eight hours on account of atelectasis, one lung never having expanded at all.

The uterus was larger than normal for the stage of pregnancy (thirty-fifth week) and the uterine muscle was 3·75 centimetres (one and a half inches) thick in the middle of the incision. In view of the theoretical objections to the administration of pressor principles to a diabetic, "Pitocin" was given to ensure proper uterine contraction during the operation.

After operation the mother's condition gave rise to no anxiety. Saline solution with glucose and insulin were available for intravenous injection, but no indication arose. The amount of insulin required to keep the urine sugar-free rapidly fell, being only ten units *per diem* on the second and third days, and five units on the fourth and fifth days. After that her urinary condition was normal, except for an occasional trace of sugar and ketone bodies. From the seventh day after operation her condition was normal in every way; she was allowed out of bed on the seventeenth day and was discharged from hospital on the twenty-first day.

This case is recorded because of the following points of interest: (i) the excessive size and the apparently accelerated development of the fœtus, due to the mother's abnormal carbohydrate metabolism; (ii) the excessive size of the uterus; (iii) the defective function of the child's respiratory apparatus; (iv) the rapid disappearance of the maternal disorder of metabolism after the removal of the fœtus; (v) the necessity for insulin in such a case to enable the mother to assimilate diet adequate for herself and the fœtus.

FOREIGN BODY IN THE BRONCHUS, WITH VERY SLIGHT RESPIRATORY EMBARRASSMENT.

By R. H. Bettington, B.M., B.Ch. (Oxon), F.R.A.C.S., D.L.O.,
Sydney.

I report this case to show how true is Chevalier Jackson's dictum that in any persistent lung infection for which no reason can be found, a foreign body should be suspected.

Clinical History.

On January 23, 1939, a baby, eight months old, was seen by its mother to fall forward on a gravel path, and she noticed the child give a slight cough. Inspection of the mouth brought to light two pieces of gravel. Being a sensible woman, she took the child to her medical attendant, who submitted it to radiological examination. He found an opaque body in the region of the opening of the right bronchus. The respirations were slightly raised and some moist sounds were present low on the right side.

The child was sent down to Sydney that night, and on January 24, 1939, another skiagram was taken. An opaque body was found in the same situation as described above.

On January 25, 1939, the child was given 5·6 cubic centimetres (one and a half drachms) of paraldehyde and sufficient narcosis was reached to allow of a sucker being introduced into the trachea. The child was then inverted and shaken, but without result. I then attempted to pass a four-millimetre "Negus" bronchoscope, but found too much laryngeal resistance; so the child was given a few breaths of ether. The bronchoscope was then passed with ease and a foreign body was seen just below the opening of the right bronchus. It was of such a shape that it was upright in the bronchus, with a good airway on each side. It was thus easily gripped with forceps and withdrawn, and was found to be a piece of gravel, rectangular in shape, measuring eight millimetres by five millimetres by three millimetres. The temperature was elevated for a day or two, and then the child went home, apparently none the worse.

Acknowledgements.

I am indebted to Dr. A. Branch, Dr. J. G. Edwards and Dr. V. Goldrick for cooperation in this case.

CONGENITAL ABNORMALITIES OF THE HEART: A DESCRIPTION OF TWO UNCOMMON VARIETIES.[1]

By Isadore Brodsky,
Sydney.

The facts of congenital abnormalities of the heart are of perennial interest, and anatomical features, even in the absence of theories of the mode of production of the abnormalities, are worthy of record. In this contribution two hearts, one in which the pulmonary veins open into the coronary sinus, and the other revealing an abnormal relation of the arterial trunks to the ventricles, are described. Unfortunately, no clinical notes are available, a fact which is regretted but which is unavoidable.

The specimens were recently donated to the department of pathology by the Renwick Hospital for Infants. Their numbers in the catalogue of the department are, respectively, 1759 and 1760, and reference to those numbers will be made in the following description.

Specimen Number 1759.

The heart is that of an infant.

The right ventricle overshadows the other compartments. The walls are moderately hypertrophied and constitute practically the whole of the anterior surface of the heart. The anterior longitudinal sulcus just fails to form the left lateral margin of the heart. The cavity of the ventricle is greater than normal, and its capacity is roughly five times larger than that of the left ventricle. All papillary muscles appear normal, and the cusps of the tricuspid valve consist of a large anterior and posterior cusp, and a small cusp at the postero-lateral angle of the opening. A capacious infundibulum is succeeded by the bulb of the pulmonary artery, whose external diameter is 1·1 centimetres at its widest part. Above the bulb the valvular elements exhibit no macroscopic abnormality. Distal to them, the *ductus arteriosus* connects the right pulmonary artery with the arch of the aorta, beyond the point where the left subclavian artery leaves the parent trunk. The diameter of the ductus, which is 3·0 millimetres, is equal to the diameter of the trunk of the innominate artery, but is imperforate.

From the anterior aspect, the presence of the left ventricle is indicated by a narrow rim of tissue lateral to the anterior longitudinal sulcus. A better estimate of its dimensions is formed from a lateral inspection; its size in relation to the right ventricle has been recorded. The walls are normal in thickness and the papillary muscles and mitral valve lack any abnormal features. The girth of the aorta is diminished, being about half that of the pulmonary artery; in position and constitution it displays no abnormality, except that the branches arise from the arch *en échelon* and a trifle proximal to the usual point of departure from the parent trunk.

[1] This investigation was carried out at the department of pathology, University of Sydney, with the aid of a grant from the National Health and Medical Research Council.

Both the left atrium and the left auricle are small. The pulmonary veins fail to communicate with the atrium, which receives blood from the right atrium by way of the *foramen ovale*, the latter being 3·0 millimetres in width.

FIGURE I.

The heart (Specimen 1759) from the anterior aspect. The horizontal probes pass from the left pulmonary veins through the coronary sinus into the right atrium.

As compared with normal, the cavity of the right atrium is dilated and the walls are slightly thicker. The

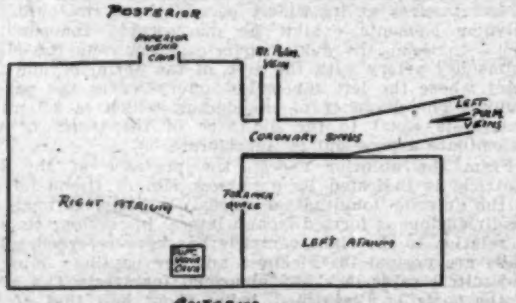

FIGURE II.

Diagram showing the relation of the pulmonary veins to the coronary sinus and the atria. The structures are being viewed from the superior aspect.

venæ cavæ open in normal situations. Conspicuous at the lower margin of the opening of the inferior *vena cava* is the Eustachian valve; this projects over a large recess, which is the abnormally wide mouth of the coronary sinus, measuring 1·0 centimetre in diameter. Into the coronary sinus the right and left pulmonary veins discharge their contents, the right opening almost at right angles to the cavity of the sinus. The left pulmonary veins join at the left lateral end of the sinus and are separated from the left atrium by a thin membranous partition. The auricle appears normal.

Specimen Number 1760.

The specimen consists of approximately the upper two-thirds of an infant's heart.

The right ventricle is uniformly hypertrophied. There is no abnormality of the papillary muscles or of the cusps of the tricuspid valve which guards a normally placed venous inlet. The pulmonary artery leaves the ventricle at the normal site, and its valves are normal in number and position. The stem is shorter and thicker than the aorta. In the *conus arteriosus* blood may be received not only from the right ventricle, but also from the left ventricle, the interventricular septum being deficient at its upper end. Prolongation of the general line of the

FIGURE III.

The heart (Specimen 1760) from the anterior aspect.

interventricular septum, in addition to separating the two ventricles, would cause the pulmonary artery to arise from the left ventricle. The aortic vestibule and trunk lie to the right and posterior to the pulmonary artery, and the proximal opening of the aorta is directly over the right ventricle, from which it receives blood. No direct connexion with the left ventricle is achieved. The aorta contains the normal complement of valves, which

are placed in their customary positions, and above them the mouths of the coronary arteries are visible. From the arch of the aorta the great vessels of the head and neck arise. The *ductus arteriosus* is pervious and branches from the main pulmonary trunk.

The left ventricle is larger than its fellow, and its walls are hypertrophied to a pronounced degree. The muscles attached to the cusps of the mitral valve are normal, and the valve fringes a normal opening into the left atrium. Blood from this ventricle has access to the pulmonary artery, though the valves of the latter are obliquely turned toward the right rather than toward the left ventricle.

The left atrium and auricle are normal. The atrium receives the pulmonary veins. The *foramen ovale* is not patent.

Both the right atrium and the right auricle are moderately distended. The superior *vena cava* is normally situated, while the inferior *vena cava* departs from normal in only one respect, in that a webbing composed of fine white cords (Chiari network) stretches across the vent on the atrial side.

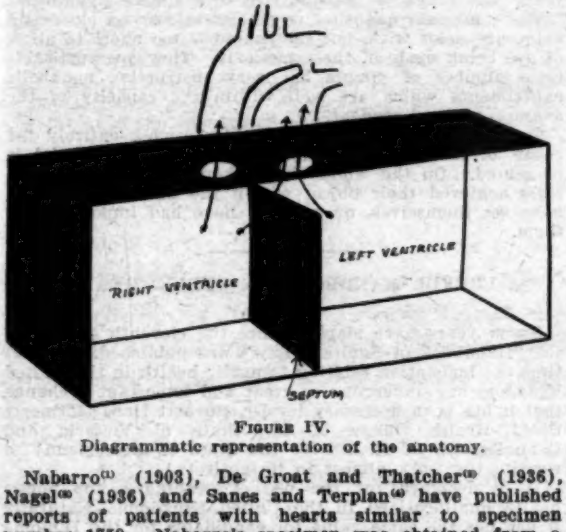

FIGURE IV.
Diagrammatic representation of the anatomy.

Nabarro[1] (1903), De Groat and Thatcher[2] (1936), Nagel[3] (1936) and Sanes and Terplan[4] have published reports of patients with hearts similar to specimen number 1759. Nabarro's specimen was obtained from a patient aged five and a half months, De Groat and Thatcher's patient lived nearly two and a half years, Nagel's patient survived for seven weeks, and the patient described by Sanes and Terplan, five months nineteen days. In her "Atlas of Congenital Cardiac Disease" Abbott[5] (1936) refers to an unreported specimen. The systemic circulation in each case depended on blood received from the right atrium through the *foramen ovale*.

Discussion.

An exactly parallel specimen to number 1760 cannot be found in the available literature. Abbott[6] (1927) mentioned that in three of Theremin's cases and in those of Lees and Rheiner, the pulmonary artery rose above a defect in the septum, and the aorta from the infundibulum of the right ventricle.

In the case under review the aorta has been transposed and arises from the right ventricle, and the pulmonary artery, though its valves are arranged over the left ventricle, is enabled to transmit blood from both ventricles because of a deficiency in the upper end of the interventricular septum.

From the standpoint of Vierordt's practical classification, as adopted by Abbott,[6] this specimen is not an example of "complete", "corrected" or "partial" transposition. ("Complete" transposition refers to the cases in which the vessels are transposed in relation to the ventricles; the transposition is "corrected" when the relation of the vessels to each other is altered, though they originate from the proper ventricles; "partial" transposition is that in which both vessels arise in a reversed relationship from the same ventricle or from a common ventricle.)

What course would the blood pursue in this case? Antenatally, the sequence of events would be the following. (i) Blood would pour into the right atrium and then be directed into the left atrium by way of the *foramen ovale*. The blood received in the left ventricle from the left atrium would then have access to the pulmonary artery, the subsequent distribution being to the aorta, through the *ductus arteriosus*, and also to the lungs. (ii) Some of the blood from the right atrium would pass into the right ventricle, whence it would be discharged into the aorta and the pulmonary artery.

In both (i) and (ii) the presence of the interventricular defect would permit a possible admixing of blood, not only in the ventricles, but also in the aorta.

Postnatally the blood would pursue the following course. On its reception in the right atrium the venous flow would be squeezed into the right ventricle. Three streams would then be dispatched, (*a*) to the aorta, (*b*) to the pulmonary artery, and (*c*) to the left ventricle. Oxygenated blood from the lungs would return to the left atrium, pass into the left ventricle and then into the pulmonary artery, after which it would feed the descending aorta (through the *ductus arteriosus*) and the lungs. A little systemic blood would cross the interventricular spur; thus the blood supply to the head, neck, heart muscle and upper extremities would be mainly of a venous character.

In this discussion it has been assumed that the *foramen ovale* was impervious immediately after birth.

Acknowledgement.

I wish to record my thanks to Mr. Woodward Smith for the photographs.

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Reviews.

A BIOCHEMICAL SURVEY OF LIFE.

It is patently consistent with the modern outlook that distinguished scientists and research workers should occasionally condescend to leave the inaccessible heights of their exclusive spheres and come down to the level of the common man. Rather than emulate the extraordinary habits of the Laputians so humorously and satirically described by Jonathan Swift in "Gulliver's Travels", two eminent doctors of science from Edinburgh have gone to some pains to give the general reader a small insight into the hidden mysteries of the biochemist's laboratory.

It is obvious that in the production of this compact volume, popularly designated "The Stuff We're Made Of",[1] the authors have entered upon their task well-fortified in the way of scholarship, literary craftsmanship and a profound knowledge of the subject. But it may be open to question whether the people they are seeking to enlighten will be capable of assimilating material so complex in constitution without its being offered to them

[1] "The Stuff We're Made Of", by W. O. Kermack, M.A., D.Sc., LL.D., and P. Eggleton, D.Sc.; 1938. London: Edward Arnold and Company. Crown 8vo, pp. 350, with illustrations. Price: 7s. 6d. net.

in a predigested form. Some chapters may not prove highly entertaining even to the medical practitioner; but a careful consideration of the book as a whole will most assuredly widen his scientific horizon and will stimulate his interest in the remarkable progress of biochemical research which is steadily increasing the scope and importance of practical medicine.

Many of us are apt to think of the living cell as the most elementary unit in our biological cosmos; and to regard atoms, electrons and molecules as belonging to a rather nebulous conception of matter. We are inclined to believe that vitamins, hormones and enzymes cannot well be defined as actual chemical entities with a known constitution; that they are more or less hypothetical substances ingeniously visualized by physiologists in order to explain away the meaning of abstruse phenomena commonly associated with the metabolism and reproduction of living cells. It is in this "No Man's Land" between the organic and the inorganic worlds that the biochemist is in his element. Here animate and inanimate particles are incapable of differentiation. But patient and laborious investigation, supported by the physical sciences on the one hand and the biological sciences on the other, is gradually bringing order out of chaos. It is quite impossible to estimate the probable influence which future biochemical discoveries may exert upon the destiny of the human race either for the betterment, or (if we heed the warnings of H. G. Wells) for the ultimate destruction, of civilization.

The reader will obtain a new perspective from the careful perusal of these philosophical pages and will appreciate still more the tremendous debt that humanity already owes to the unobtrusive worker in this comparatively new field of scientific research.

HYGIENE AND MIDDLE AGE.

IN "Health in Middle Life" Dr. Ethel Browning has written a small book intended to be helpful to men and women passing through middle life, assisting them to grow old gracefully.[1] She aims at getting rid of the dangers of half-knowledge and tries to help her readers to make mental and emotional adjustments to their difficulties. A sense of proportion she regards as most important to a successful climacteric. Adjustment is the keyword.

The book may well be helpful to doctors preparing talks to the laity on such subjects, and may be lent to selected patients.

Much attention is given to the action of the endocrine glands in both men and women. The diet is also given in much detail. Instructions are given for exercise, for relaxation and for the development of new interests at and after the time of retirement.

The particular physical and emotional troubles which lie in wait for the unhealthy or the unwise at that time are related quite fully enough; so that the emphasis on ill-health rather than on health is difficult to avoid. The book may be regarded as a moderately successful application of hygiene and common sense to the management of middle age.

PRACTICAL BIOCHEMISTRY.

IN preparing the second edition of their "Practical Physiological Chemistry", Professor Wishart and his collaborators have modified their compact little manual slightly.[2] A more robust format has been adopted, no doubt as a result of experience of the effect of laboratory use on paper covers. Line illustrations of common urinary and other crystals and a coloured plate of absorption spectra of blood pigments have been added. The useful blank interpages have been retained.

The course is designed for twenty two-hour practical periods. The conventional qualitative examinations of materials of biological importance are adequately, if briefly, dealt with. One or two points in the section on proteins have not received the revision which they needed. The whole question of protein precipitation, including heat coagulation, has not withstood the rather drastically compressed treatment as well as other sections. The different types of protein precipitation are by no means clearly distinguished. In the table of classification of proteins the use of the term "native" proteins for albumins and globulins may lead to confusion. The term "native" is now usually employed to distinguish unchanged from denaturated proteins.

The treatment of digestion and enzyme action could also be expanded with advantage. Compression has led to simplification which is not wholly justified. For example, regurgitation from the duodenum is given as the sole explanation of the course of the curve of gastric acidity. Unfortunately much work within the last ten years has failed to substantiate this simple hypothesis.

The commoner quantitative determinations on blood and urine are dealt with, but the course is too short to allow of use being made of these methods. They are applicable to a number of simple but very instructive metabolic experiments which are well within the capacity of the average medical student.

The shortcomings of the book are almost entirely the result of the compressed form in which the material is presented. On the whole the authors have very successfully achieved their object within the limits which they have set themselves, or perhaps, have had imposed upon them.

A USEFUL HANDBOOK ON PUBLIC HEALTH.

SEVEN years have elapsed since the eleventh edition of the "Handbook of Sanitary Law" was published. In that time the legislation relating to public health in the United Kingdom has undergone so great and important a change that it has been necessary for Dr. Burnett Ham (formerly Chief Health Officer of the State of Victoria and Commissioner of Health for the State of Queensland) to rewrite the work almost in its entirety.[1]

It is designed for the use of candidates for public health qualifications in the United Kingdom. Naturally it has not the same value for candidates for the Diploma of Public Health or the Royal Sanitary Institute certificate in this country; for although our health legislation may be based on the same principles, it differs entirely in detail, not only between Australian States and the United Kingdom, but between one State and another. The work, however, will be of use in Australia to those interested in public health, such as full-time health officers, members of Parliament, members of townplanning commissions and the like, who desire to familiarize themselves with the trend of recent legislation overseas without the necessity of perusing in detail a large number of Acts. It summarizes the provisions of over forty Acts of Parliament, twelve sets of regulations and one Order-in-Council, dealing with the public health in every one of its aspects. All the essential features which have characterized previous editions have been maintained in this twelfth edition, and in the reduction of the statutory text to a simpler form care has been taken to ensure accuracy consistent with conciseness. This has been achieved in twelve chapters and one appendix in 356 octavo pages. The volume is compact and light. It is printed on good quality paper, the type is clear and the indexing complete. It can be confidently recommended as a handy work of reference to all public health workers interested in the subject.

[1] "Health in Middle Life", by E. Browning, M.D.; 1938. London: Rich and Cowan Limited. Foolscap 8vo, pp. 249. Price: 3s. 6d. net.

[2] "Practical Physiological Chemistry for Medical Students", by G. M. Wishart, D. P. Cuthbertson and J. W. Chambers; Second Edition, revised; 1938. Glasgow: John Smith and Son (Glasgow) Limited. Crown 8vo, pp. 128. Price: 4s. net.

[1] "Handbook of Sanitary Law for the Use of Candidates for Public Health Qualifications", by B. B. Ham, M.D., D.P.H.; Twelfth Edition; 1938. London: H. K. Lewis and Company Limited. Foolscap 8vo, pp. 378. Price: 7s. 6d. net.

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Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

DUTY, SERVICE AND UNITY.

THE review by Dr. George Barron in his president's address of the early occupants of the office of president of the New South Wales Branch of the British Medical Association will not be only be of interest to historically minded members of the Association in Australia, but should also be a stimulus to those who have any care for the future of the profession. The men in Dr. Barron's list of presidents were distinguished in various spheres, some of them in many spheres. Search in the archives of other Australian Branches would reveal records of men of like distinction. These followed the light as they saw it and, being men of probity, ability and audacity, built on a secure foundation. The edifice that they built is handed on to the present generation to be cherished and honoured and withal to be extended in usefulness. The science of medicine will never be complete, nor will society cease from troubling and be at rest; and so the coping stone of this building will never be set, however greatly we may honour and cherish it and its traditions.

In seconding a vote of thanks to Dr. Barron for his address, Dr. H. R. R. Grieve said that Dr. Barron's words were a call to duty, a call to service and a call to unity. At the present time the call to duty and service is probably greater than it has been for some years past. Medical practitioners in Australia have been faced with problems resulting from changes in society and from governmental action. The attempts to find a solution are fresh in the minds of all members of the Association, and the whole profession stands indebted to the members of its councils and committees who have given freely of time and ability in service to the common good. The call to service continues, for whatever be the result of recent negotiations, the welfare of the medical profession depends on its willingness to spend and be spent in service of its members and of the community. This service must be given of a free heart and in full measure; it must not be a service of self-seeking. If the members of the British Medical Association continue to respond to the call of service, as we believe they will, the call to unity will scarcely be necessary. But in many counsels there is diversity of opinion and disputants are not always able to see the point of view of their opponents. Even if they cannot go so far as this, those who uphold one view can always allow to those of another opinion honesty of purpose and freedom from guile. In this way divergent views can often be reconciled. Union then becomes possible.

In all Australian Branch discussions on medico-political matters the highest ideal of service must be kept in the forefront and every branch of the profession must take part. In other words, the Australian Branches must represent the medical profession as a "whole body fitly joined together". Men with new ideas must be welcomed in counsels and discussions, and those who have spent years in the common cause must be given an opportunity of using their experience. History in medico-political affairs, like all other history, repeats itself in surprising fashion; and what is difficult to the new-comer may be simple to the older member. This will be clear to anyone who cares to study the history of any of the Branches. The conclusion of the whole matter is that no clash of sectional interests must be allowed to take from the strength of the whole body. "The body is not one member, but many. If the foot shall say, Because I am not the hand, I am not of the body; is it therefore not of the body? But now *are they* many members, yet

but one body . . . That there should be no schism in the body; but that the members should have the same care one for another."

Current Comment.

THE PSYCHOANALYTIC TREATMENT OF CHRONIC ADDICTION TO ALCOHOL.

Addiction to alcohol is a disease, not a habit. It is primarily a psychiatric problem calling for a deep understanding of the dynamics of behaviour; for no amount of punishment, moralizing or preaching will favourably influence the underlying personality disorder of which the addiction is the main symptom. It amounts to a major sociological problem in many countries; but until the psychiatrist can take the lead it is unlikely to receive the attention it merits. The psychiatrist may feel at home when confronted by patients suffering from *delirium tremens,* Korsakow's psychosis or pseudoparesis, but be quite unable to cure a simple case of addiction to alcohol which has not as yet given rise to any of these psychotic complications. More than ordinary difficulties may confront him. Interference by relatives, blatantly advertised yet bogus "cures", inadequate hospital facilities, legal restrictions and financial considerations often conspire to defeat the most laudable and persistent therapeutic efforts. Not the least of these adverse factors are the patients themselves, as Robert P. Knight has shown in his article[1] on the psychoanalytic treatment of chronic addiction to alcohol, wherein he gives his experience of treating twenty chronic addicts by psychoanalysis within a sanatorium.

Knight's thesis is that no alcohol addict is normally adjusted even when sober, and the value of analysis lies in its being able to discover the individual aspects of the problem and their relation to environmental situations. He recognizes three types of alcoholic addicts. The first are the "essential alcoholics". Such cases arise as the result of intrapsychic conflicts engendered in early life by parental influence. They are frequently the sons of dominant and over-protective mothers and alternately stern and indulgent fathers. From childhood they grow up with emotional maladjustments and fixations at infantile levels. Drinking solves the conflict between passive feminine wishes and masculine strivings. Addiction commences early in life and the patients show a characteristic psychopathic reaction to life's problems and an inability to make any sustained effort in adult undertakings. The prognosis in such cases is bad. The patients are seldom able to face up to the rigors of the treatment. Feelings of dependency defeat the wish to be cured. For such patients reality is too harsh to be acceptable; the cloak of alcoholism is too comfortable. And no matter how diligent and persuasive the therapist, he is generally unable to offer them any satisfaction in sobriety comparable with the pseudo-enjoyment of the narcotic refuge.

More hopeful in outlook are patients who fall into the second of Knight's groups. These he calls "reactive alcoholics". In these the personality is better integrated. These patients are not so severely handicapped by emotional bondage. They have a better record of personal achievement and are made of "sterner stuff" than the essential alcoholics. Their attitude to treatment is hopeful and more determined. Their addiction is characteristically periodical and is the result of definite precipitating stress. The prognosis for such patients is generally favourable. "Symptomatic alcoholics" which constitute Knight's third group, are patients who are suffering from some major psychotic disorder, in which alcoholism has become an incidental symptom. Alcoholism may mask the otherwise characteristic clinical picture of schizophrenia, melancholia or an organic psychosis. The prognosis depends upon the nature and the response to treatment of this underlying psychotic state. It is believed that patients in the first two groups are best treated by psychoanalysis within an institution. Details of nursing and administration, restrictions, indulgences, outlets for aggressive drives and attitudes calculated to allay anxieties are revised periodically to suit the needs of the patient. The analysis follows largely along classical lines, depending mainly in the wise handling of the transference, the ability to recognize in the behaviour of the patient any evidence of hostility, distrust or affection, and to make explanations and interpretations at opportune times. There is no magic in psychoanalysis. The outcome depends upon the patient's continued desire to be cured; that is, to be rid of his conflicts and reeducated to the point at which he can successfully handle reality situations. In favourable cases a stage is soon reached when the patient may be trusted to go back to his home surroundings and continue his analysis without institutional supervision. This change may prove a crucial test of the treatment. Old associations may reawaken old desires and the cocktail claim the new convert to abstinence. Treatment fails, according to Knight, for two reasons: the flagging desire for help in the arduous process of working out one's conflicts which is bound up with the natural resentment of having to relinquish childish satisfactions, and the frequent interference of relatives, who seem more inclined to meddle with the treatment of alcoholics than in most other psychiatric conditions. Disillusionment born of past failures and an array of broken promises seem no deterrent to relatives, who are always willing to believe the patient is fully repentent and ready for another try.

Knight's results are anything but brilliant; but the scheme of treatment which he lays down, occupying from eighteen months to three years, is probably the most satisfactory way of handling a psychiatric problem, the outlook for which in the present state of our knowledge is always serious and often hopeless.

[1] *The Journal of the American Medical Association,* October 15, 1938, page 1442.

RENAL ISCHÆMIA AND HYPERTENSION.

Only during the last few years has any precise knowledge of the relationship between renal disease and hypertension been acquired. Many attempts have been made to produce hypertension in laboratory animals by operative procedures on the kidneys, such as ligation of branches of the renal artery or removal of varying amounts of renal tissue. None of these procedures, however, had any constant effect on the blood pressure, and it was not until 1934 that a reliable method was developed of producing experimental renal hypertension. In that year Goldblatt and his colleagues succeeded in producing persistent hypertension in dogs by restricting the blood supply to the kidneys with clamps applied to the renal arteries. This achievement immediately opened up a large and promising field of inquiry, which has since claimed the attention of many workers. Among these are E. B. Verney and Marthe Vogt,[1] who carried out experiments on renal hypertension in dogs, the results of which confirm and extend Goldblatt's original observations.

Verney and Vogt's blood pressure measurements were made by compression of an "externalized" loop of carotid artery with a specially made miniature sphygmomanometer cuff. Reliable figures were obtained, provided that the carotid sinus on that side had been previously denervated. Naturally this method disturbed the animals much less than direct manometric measurements from the femoral artery. To restrict the renal blood supply, Verney and Vogt partially occluded one or both renal arteries with silver clips. In nearly all cases a rise of blood pressure occurred, beginning a few hours after the operation and persisting for several weeks or months until the animal was killed. The degree of hypertension varied from 60 to 120 millimetres of mercury above the normal pressure; it was much greater in those animals with both renal arteries narrowed than in the unilaterally treated animals. The animals appeared to be perfectly healthy, with no loss of appetite or change in behaviour, and blood analysis showed the non-protein nitrogen content to be normal. At autopsy the affected kidneys were smaller than at the time of operation, but otherwise were normal macroscopically. In those animals which failed to develop hypertension after partial occlusion of the renal arteries, the authors showed that an efficient collateral blood supply had arisen from the perinephric tissues and that the blood pressure would rise as in the other animals if these vessels were tied.

The bulk of Verney and Vogt's experiments was concerned with the elucidation of the mechanism which produces hypertension in these animals. That retention of waste products by failing kidneys plays no part in its production was shown by the observation that removal of the ischæmic kidney from unilaterally treated dogs caused the blood pressure to fall abruptly to normal, in spite of the reduction in the amount of functional kidney tissue; furthermore, removal of renal tissue from normal dogs, even to the point at which signs of renal failure were produced, did not cause hypertension. Total nephrectomy was performed in a number of dogs without any resultant rise in blood pressure. The authors concluded, therefore, that the presence of ischæmic renal tissue was the condition essential for the development of experimental hypertension. In what way does a starved area of kidney tissue cause a rise in blood pressure? Verney and Vogt showed that it is not a reflex phenomenon actuated by afferent impulses travelling via the renal nerves, because restriction of the renal blood flow caused hypertension even in dogs whose kidneys had been previously denervated. Hence there is no doubt that the cause of experimental renal hypertension is a chemical substance liberated by ischæmic renal tissue. Direct evidence on this point was obtained by Verney and Vogt, who perfused a length of intestine from a heart-lung-kidney preparation, of which one kidney was ischæmic and could be introduced into and out of the circulation at will. Whenever this kidney was included in the circulation, vasoconstriction occurred in the perfused gut. In another series of experiments excision of the abdominal and thoracic sympathetic chains was performed in several dogs. The blood pressure after this procedure was low and rather unstable, but otherwise the animals were fairly normal. Restriction of the renal blood flow in these animals caused hypertension as in animals with their sympathetic system intact, an observation which demonstrates that the "hypertensive substance" acts peripherally and not on the central vasomotor mechanism.

That the degree of hypertension is determined by the load on the ischæmic renal tissue was shown by Verney and Vogt in several ways. Firstly, if one kidney was removed from a hypertensive dog with bilateral renal ischæmia, the blood pressure became raised still further. Secondly, a similar aggravation of hypertension occurred if the sound kidney was removed from a unilaterally treated animal. Thirdly, a high salt and protein diet produced an increase in blood pressure in hypertensive dogs, though it had no effect on the blood pressure of normal animals or of animals suffering from renal deficiency without ischæmia, in spite of the increase in plasma non-protein nitrogen. In dogs with very severe restriction of the renal blood supply, especially if a high protein diet was given, a syndrome appeared very like the uræmic syndrome in man. Verney and Vogt believe that this syndrome is caused by the same substance which is responsible for simple hypertension, though there is no direct evidence on this point.

Although at this stage the work on experimental hypertension can suggest no great advance in the treatment of similar conditions in the human being, nevertheless it is of great importance, because it promises to establish the pathology of these diseases on a sound experimental basis, a necessary preliminary to rational therapeutic advance.

[1] *Quarterly Journal of Experimental Physiology*, Volume XXVIII, Number 3, 1938, page 253.

Abstracts from Current Medical Literature.

THERAPEUTICS.

Manganese Poisoning.

E. W. Baader (*Archives des maladies professionnelles*, May-June, 1938) discusses manganese poisoning. Intoxication occurs mainly in the mines and in the manufacture of iron and steel, which contain 10% to 30% of manganese; but it has also been found in various industries, such as glass, colour, alloy and dry battery manufacture and in pharmacy. It is a rare disorder, commencing with lassitude and drowsiness or loss of balance, a festinant gait, spasticity or steppage gait. Tremors, ataxia and slurring speech and a mask-like face are noted, followed by spasmodic laughter or tears. Only four autopsies were performed. These showed atrophy and degeneration of cells in the cortex and grey matter of the basal ganglia. Graves's disease, erythrocythæmia, fever and violent cramps have been recorded. The mental state is unaffected. Cirrhosis of the liver and pneumonia have also been mentioned as probable results of manganese intoxication.

Sulphanilamide Therapy and Toxicity.

E. G. Bannick *et alii* (*The Journal of the American Medical Association*, August 27, 1938) amplify their earlier reports on sulphanilamide in particular regard to dosage and toxic effects. Experiences with "Uleron" and "Neoprontosil" are also recorded. Sulphanilamide is of definite value in diseases resulting from infection with hæmolytic streptococci, meningococci, gonococci and all urinary tract infections, with the exception of those due to the *Streptococcus fæcalis*; it is unsatisfactory in staphylococcal infections, rheumatism and *Streptococcus viridans* infection. Exact dosage is still unsettled, depending largely on the type and severity of the illness. The studies of Long suggest that the optimum blood concentration is 10 milligrammes per 100 cubic centimetres of blood; the authors are of the opinion that this concentration should be attained quickly and maintained until there is clinical improvement or until toxic symptoms develop. It is also desirable to maintain a satisfactory blood concentration by uniform intervals between doses. Mild toxic manifestations are malaise, anorexia, nausea, vertigo and tinnitus; more severe reactions are cyanosis, abdominal cramps, diarrhœa, cutaneous lesions and fever. The serious symptoms are leucopenia, acute anæmia with crisis, jaundice and shock. The examination of the blood is the best guide to the degree of toxicity. It was found that a lower blood concentration was necessary when dimethyl-di-sulphanilamide or "Uleron" was used, 1·9 milligrammes being adequate, and maintained by the oral administration of an average daily dose of 4·0 grammes given in five or six doses. Toxic symptoms are less frequent and often patients who cannot take sulphanilamide without toxic manifestations can tolerate "Uleron". Peripheral neuritis, affecting chiefly the lower limbs, is a complication of treatment, and it may fail to recover completely. The incidence is low. "Neoprontosil" is discussed also; the oral administration is of greater usefulness than the parenteral, as excretion is slower. There are fewer toxic reactions and the clinical results are satisfactory. Its chief value at present is its usefulness as an alternative to sulphanilamide.

Lead Poisoning.

E. C. Vigliani (*Archives des maladies professionelles*, July-August, 1938) describes new methods of diagnosing saturnism. It was found that in the metabolism of porphyrine lead poisoning caused an abnormality. Porphyrine appeared in the fæces and urine in excess of the normal as early as the fifth day in poisoning by lead, whereas other changes in the blood did not appear until the tenth day or later. Another new test was that by which the presence of lead in the blood was determined. The author did this by burning the ash of a little blood and examining the spectrum for lead, which gave characteristic appearances. In order to determine the amount of lead in the blood more complex methods were used; the rays produced in the spectroscope by lead were compared with those of molybdenum. The measurement of the intensity of these rays on the spectrum was plotted and the amount of lead present in the blood determined.

Thymol in Actinomycosis.

H. C. Fang (*Chinese Medical Journal*, November, 1938) reports a case of actinomycosis of the jaw and reviews briefly the present status of therapy. Iodides, iodine, "Yatren", copper sulphate, "Salvarsan", silver nitrate, methylene blue, vaccines *et cetera*, have all proved useful in some cases but not others. Röntgen therapy has been successfully employed in various instances and seems to be of most benefit when the lesion is localized to the head and neck, as the high dosage necessary to be successful is likely to damage other structures in thoracic and abdominal infections. Surgery is useful as a complete cure only if it is radical. There are obvious limitations; as a supportive measure, however, surgery is indispensable in all forms of treatment. The laying open of sinuses, the evacuation of pus pockets and the removal of dead tissue are essential steps in therapy. Meyers had reported success in a case of actinomycosis with the use of thymol, and the author used this substance in his case after surgery had rendered accessible the active portions of the lesion. One gramme of thymol in half-gramme capsules was given before breakfast every other day until cure was complete, some four months later. In addition, local applications of 10% thymol in olive oil were applied in the form of compresses. Various other forms of treatment had been of no avail in this case; the internal administration and the local application of thymol, together with such surgical measures as mentioned above, were successful in bringing about a cure. It is thought that thymol exerts a direct fungicidal action on the actinomyces. It is possible to produce a sufficiently high concentration in the blood to be effective; up to two grammes every other day can be given. The local applications will enhance the benefit of the internally administered drug; a strength of 20% thymol in olive oil is permissible. Occasionally the patient may develop gastro-intestinal disturbances, such as anorexia and loose motions. The temporary withdrawal of the drug will overcome this difficulty.

Calcium Cevitamate.

S. L. Ruskin (*The American Journal of Digestive Diseases*, September, 1938) found, during the course of chemical studies of vitamin *C*, that cevitamic acid dissolved the insoluble calcium carbonate with almost equal rapidity and completeness as strong hydrochloric acid. This solubility suggested the possibility of a strong ionizing influence on calcium, and experiments were carried out to determine this point; results are given which prove that calcium cevitamate is highly soluble and gives a high percentage of ionized diffusible calcium. This substance is a white powder, readily soluble, and it is thus available in physiological solutions up to 40%. It is non-irritating on injection, either subcutaneously, intramuscularly or intravenously; it is well tolerated by the gastro-intestinal tract. There is a remarkable parallelism of action between calcium and cevitamic acid in bone metabolism, hæmorrhagic diathesis, cell membrane permeability and detoxicating action; so close is this action that their functions can be interchanged. The chemical properties of vitamin *C* would indicate that it is the stabilizing agent for calcium in the body, participating in absorption and maintenance of the ionized calcium in serum calcium; the advantages of the combination of these two substances in one chemical combination are obvious.

NEUROLOGY AND PSYCHIATRY.

Surgery in Psychiatry.

James W. Watts and Walter Freeman (*The Journal of Nervous and Mental Disease*, November, 1938) report their experiences with prefrontal lobotomy in psychotic patients, following the method first introduced

by Egas Moniz. This writer obtained the best results in cases of agitated depression, in which, he believed, fixed patterns of response were showing a tendency to perpetuate themselves to the detriment of the patient. The worst results were among patients of the schizophrenic type. The surgical technique is described, the authors having modified Moniz's original leucotome to permit the injection of thorium dioxide solution without removal of the instrument from the brain. This leucotome also has a double instead of a single blade. Regardless of the diagnosis, patients were chosen showing a particular symptomatology — mental tension, agitation and anxiety. Only such patients were chosen, however, whose symptoms seemed to indicate an unfavourable prognosis and in whom all other recognizable forms of treatment had been tried without improvement. The report covers twenty such cases. Of the patients, 15% were greatly improved and an additional 50% were moderately improved. The operative mortality was 5%. After operation there was a reduction of mental tension, depression and agitation; suicidal ideas disappeared and hallucinations gradually disappeared. Directly after operation the patients become unresponsive and uncommunicative; they speak only when spoken to. There are an emotional flattening, diminished spontaneity, lack of attention and indifference. Transient perseveration of speech and disorientation are reported. Numerous transient somatic disturbances are reported, particularly in the bladder.

Prostigmin in Myasthenia Gravis.

M. TARLAU (*The Journal of Nervous and Mental Disease*, September, 1938) reports two patients with *myasthenia gravis* treated with prostigmin. Reference is made to the literature of this condition and to other reports upon the use of prostigmin, which was first introduced as a drug of value in post-operative ileus. The first patient in this report was a Jewess, aged twenty-two years, who came up for investigation complaining of double vision and rapid tiring. Complaints of paroxysmal weakness were slowly progressive. There was no major psychic conflict. Muscle power was good in all muscles initially, but it tired rapidly against resistance. No pathological reflexes were elicited; gait was normal and no other neurological signs were present. No abnormality was found in the blood or cerebro-spinal fluid. A diagnosis of *myasthenia gravis* was made and the patient was given prostigmin, which caused a rapid amelioration of symptoms. The dosage varied with the patient's energy requirements. She generally took 15 milligrammes every four hours and a subcutaneous injection of 0·25 milligramme was given at those times when extra energy was required. The second patient was a coloured woman of thirty-four, who complained of weakness and inability to talk and swallow. She also suffered from diplopia and had an oculoptosis on the right side and weakness in the extremities. She responded temporarily to the administration of prostigmin in ordinary doses, but for no accountable reason developed a rapidly increasing tolerance to the drug. Finally it required a dose of 240 milligrammes to enable the patient to eat, and as larger doses produced toxic manifestations she became worse and died in her sleep. Many adjuvants were tried without avail. The author concludes that prostigmin produces better results in *myasthenia gravis* than any other drug, but so far it has shed no light upon the ætiology of this condition.

Chronic Subdural Hæmatoma.

CORNELIUS G. DYKE AND LEO M. DAVIDOFF (*Bulletin of the Neurological Institute of New York*, September, 1938) classify the twenty-four cases of chronic subdural hæmatoma which make up their study as: (i) chronic calcified hæmatoma, (ii) relapsing juvenile chronic subdural hæmatoma, (iii) chronic subdural hæmatoma following a single injury, usually occurring in adults. The article is profusely illustrated by Röntgenographic and pneumoencephalographic plates. The authors report two cases of chronic calcified hæmatoma, in which there were long-standing histories of neurological signs and symptoms. Patients with relapsing juvenile chronic subdural hæmatoma gave histories of early trauma in infancy or childhood and a history of subsequent trauma just prior to admission to hospital. On radiological examination they showed long-standing changes in the skull, and the encephalograms revealed a relatively slight shift and deformity of the ventricles. Mental changes associated with headache and vomiting but with few neurological signs were generally found with the chronic subdural hæmatoma following a single injury in adults. Radiologically there was little change in the *sella turcica*, but generally a considerable downward displacement of the pineal shadow. In the encephalograms displacement of the ventricular system was evident. The subarachnoid sulci markings were generally absent on the side of the lesion. In none of the cases reported was there any evidence of fracture of the skull.

The Cortical Dysrhythmias of Schizophrenia and Psychomotor Epilepsy.

THE electro-encephalographic studies made by F. A. Gibbs, E. L. Gibbs and W. G. Lennox (*The American Journal of Psychiatry*, September, 1938) reveal a relationship between schizophrenia and epilepsy. Far from there being a mutual antagonism between them, as Meduna has suggested, these authors find a positive relationship exemplified in the electrical activity of the cerebral cortex. They make due allowance, in summing up their evidence, for the fact that both "epilepsy" and "schizophrenia" are waste-paper basket terms, which include a wide variety of psychological reactions unrelated by any accepted ætiological pathological change. The electro-encephalographic record obtained in patients having psychomotor seizures is similar to that which may be seen in patients suffering from schizophrenia, and similar also to the records obtained from psychopathic children. The authors further found that the frequency and pattern of the waves might be influenced by physiological changes in the body. Drugs also cause alteration in the frequency of the cortical rhythms, the most valuable of these in ameliorating cerebral dysrhythmias being sodium diphenyl hydantionate ("Dilantin").

Multiple Therapy in the Psychoses.

H. H. GOLDSTEIN, E. F. DOMBROWSKI AND J. V. EDLIN (*The American Journal of Psychiatry*, September, 1938) discuss the results which various European and American workers have obtained by the use of insulin and "Cardiazol" in the treatment of schizophrenia. Their own experience with these forms of treatment goes to show that some patients who derive no benefit from insulin may recover after the use of "Cardiazol" and *vice versa*. They do not feel justified in specifying the advantages of one form of treatment over the other, but state that both forms of treatment should be available for schizophrenic patients in psychiatric hospitals, and they suggest that treatment by "Cardiazol" should be instituted first on account of its less difficult and lengthy technique, and because it is less costly than insulin. The authors are silent in regard to the benefits of a combination of both forms of treatment at the same time.

Hydration Studies in Epilepsy.

EUGENE ZISKIND, ESTHER SOMERFIELD-ZISKIND AND RUTH BOLTON (*The Journal of Nervous and Mental Disease*, January, 1939) set out to demonstrate that excessive hydration is a precipitant of epileptic convulsions. They were able by the administration of large quantities of water to produce fits in 50% of the sixteen epileptic patients selected for study. They found that blood dilution and water retention were greater in the epileptics than in non-epileptics after excessive water intake. They determined blood dilution by weighing two cubic centimetre samples of whole blood before and after desiccation. The blood chloride content was found to be diminished following excessive hydration. Such studies have a bearing on the dehydration treatment of epilepsy; and these authors, while demonstrating that excessive water intake will often but not invariably cause fits, cast considerable doubt on the value or efficacy of dehydration in the control of convulsions.

British Medical Association News.

ANNUAL MEETING.

The annual meeting of the New South Wales Branch of the British Medical Association was held at the Robert H. Todd Assembly Hall, British Medical Association House, 135, Macquarie Street, Sydney, on March 30, 1939, Dr. B. T. Edye, the President, in the chair.

Annual Report of Council.

On the motion of Dr. L. A. Dey, seconded by Professor W. K. Inglis, the annual report was received. The report is as follows.

The Council presents the following report on the work of the Branch for the year ended March 30, 1939.

Membership.

The membership of the Branch is now 1,791, as against 1,741 at the date of the last report. The additions have included 105 elections, reelections and resumptions, and 29 removals into the area of the Branch; while the losses have included 5 by resignation, 29 removals out of the area of the Branch, 31 by default in payment of subscription, and 19 by death. The losses by death were as follows: Dr. N. C. Larkins, Dr. F. G. Baur, Dr. F. Short, Dr. W. J. White, Dr. T. Farranridge, Dr. W. A. James, Dr. A. L. Kerr, Dr. H. C. E. Donovan, Dr. J. Colman Woods, Dr. L. Swinnerton Duke, Dr. W. B. Dight, Dr. C. H. E. Lawes, Dr. K. M. Whiting, Dr. E. B. M. Vance, Dr. S. Douglas, Dr. J. E. Webb, Dr. P. M. Wood, Dr. W. C. Mansfield, Dr. R. A. Muller.

Meetings.

Ten ordinary meetings of the Branch (including the annual general meeting), five extraordinary meetings and eight clinical meetings were held. The average attendance was 54. Nine of the ordinary meetings were held in conjunction with meetings of sections, namely: April 28, with the Section of Surgery and the Section of Radiology; May 26, with the Section of Pædiatrics and the Section of Pathology and Bacteriology; June 30, with the Section of Medicine and the Section of Radiology; July 28, with the Section of Neurology and Psychiatry and the Section of Obstetrics and Gynæcology; August 25, with the Section of Orthopædics and the Section of Pædiatrics; September 29, with the Section of Medicine and the Section of Oto-Rhino-Laryngology; October 27, with the Section of Obstetrics and Gynæcology and the Section of Pathology and Bacteriology; November 24, with the Section of Neurology and Psychiatry and the Section of Medicine; December 8, with the Section of Orthopædics and the Section of Surgery. The clinical meetings were held at the Royal Alexandra Hospital for Children, the Royal Prince Alfred Hospital, the Royal North Shore Hospital, the Women's Hospital (Crown Street), Lewisham Hospital, Sydney Hospital, Saint Vincent's Hospital and the Renwick Hospital for Infants. The business of the meetings included nineteen papers and addresses, numerous reports of cases, exhibits and demonstrations, and the showing of films. The extraordinary meetings held on April 21 and May 5 were convened to give members an opportunity of discussing the National Health and Pensions Insurance Bill which was before the Commonwealth Parliament. Two of the extraordinary meetings, held on April 28, were convened for the purpose of amending the Articles (Article 67) and By-Laws (By-Law 27) in regard to the formation of Special Groups, and at the fifth, held on May 26, a new By-Law (By-Law 21A) was constituted. An invitation was extended to the fifth and sixth year medical students of the University of Sydney to attend the ordinary and clinical meetings of the Branch.

Representatives.

The Branch was represented as follows:

1. Council of the British Medical Association (1938-1941): Professor R. J. A. Berry.
2. Representative Body of the British Medical Association (1938-1939): Representative, Dr. L. A. Dey. Deputy Representative, Dr. H. C. Rutherford Darling.
3. 106th annual meeting, British Medical Association, Plymouth, 1938: Delegates, Dr. H. C. Rutherford Darling, Dr. A. Muscio.
4. Federal Council of the British Medical Association in Australia: Dr. George Bell, O.B.E., Dr. W. F. Simmons.
5. National Health Insurance Committee of the Federal Council: Dr. George Bell, O.B.E., Dr. W. F. Simmons. General Practitioner Representative, Dr. H. R. R. Grieve. Co-opted, Dr. R. C. Traill.
6. Australasian Medical Publishing Company, Ltd.: Dr. T. W. Lipscomb, Dr. F. P. Sandes, Dr. A. M. Davidson, O.B.E.
7. New South Wales Post-Graduate Committee in Medicine: Professor W. K. Inglis, Dr. L. W. Dunlop.
8. Ninth Australian Cancer Conference, Sydney, April 5 to 8, 1938: Dr. F. P. Sandes.
9. Tenth Australian Cancer Conference, Wellington, New Zealand, February 14 to 18, 1939: Dr. B. T. Edye, Dr. C. F. A. de Monchaux.
10. Australian and New Zealand Association for the Advancement of Science: Professor Henry Priestley, Professor Harvey Sutton.
11. Ophthalmic Association Limited: Dr. J. A. Flynn.
12. Australian Aerial Medical Services: Representative, Dr. George Bell, O.B.E. Deputy Representative, Dr. J. G. Hunter.
13. Council of the Royal Society for the Welfare of Mothers and Babies: Sir Robert Wade, Dr. E. H. M. Stephen.
14. Council of the Bush Nursing Association (1938-1939): Dr. B. T. Edye.
15. Board of Control of the Campaign against Tuberculosis: Dr. A. S. Walker.
16. Metropolitan Hospitals Contribution Fund of New South Wales: Dr. R. V. Graham.
17. St. John Ambulance Association: Dr. B. T. Edye.
18. Council of the New South Wales Institute of Hospital Almoners: Dr. W. Vickers.
19. Executive Committee of the Council for Mental Hygiene for New South Wales: Dr. C. K. Parkinson.
20. Standards Association of Australia: (i) Institutional Supplies Committee: Dr. S. W. G. Ratcliff. (ii) Sectional Committee on Interior Illumination of Buildings: Dr. E. A. Brearley.
21. Council of Education: Dr. A. J. Collins.
22. Medical Officers' Relief Fund (Federal); Local Committee of Management for New South Wales: Dr. E. H. M. Stephen, Dr. A. M. Davidson, Dr. A. J. Collins.
23. Road Safety Council: (i) Committee for the determination of visual standards for motor drivers: Dr. R. G. Waddy. (ii) Committee for the determination of the physical capacity of motor drivers: Dr. J. Hoets.
24. Recreation and Leadership Movement: Professor Harvey Sutton, Dr. W. C. McClelland.
25. Conference on Education: New South Wales Public School Teachers' Federation: Dr. E. H. M. Stephen, Dr. A. J. Collins.
26. Police Boys' Club: Dr. B. T. Edye.

27. Department of Education: Physical Education. (i) Physical Education Advisory Committee: Professor Harvey Sutton. (ii) Pre-School and Subprimary Committee: Dr. J. Hoets, Dr. D. G. R. Vickery.

28. Medical Appointments Advisory Committee (Hospitals Commission of New South Wales): Dr. W. Vickers.

The Late Dr. C. H. E. Lawes.

The Branch has suffered by the passing of Dr. C. H. E. Lawes. He was a member of the Branch for 39 years, a member of Council for 23 years, President in 1923, Honorary Secretary 1932-1937 and Vice-President 1937-1938. In addition, he acted as representative of the Branch on the Federal Committee in 1932-1933 and was the first Secretary of the Federal Council.

The Council has placed on record its deep regret at his death and its sincere appreciation of the valuable services he rendered to the Association. The sympathy of the Branch is extended to the family.

Council.

(*a*) The attendance of members of the Council and of the standing committees was as set out in the accompanying table.

(*b*) The representatives of the Local Associations of Members, appointed on the invitation of the Council to attend the regular quarterly meetings of the Council, were as follows: Dr. R. A. Robertson (Border), Dr. C. G. Bayliss (Canterbury-Bankstown), Dr. A. M. Gledden (City), Dr. J. H. Coles (Central Southern), Dr. A. L. Bryant (Central Western), Dr. B. W. Stevenson (Eastern Suburbs), Dr. L. W. Wing (Far South Coast and Tablelands), Dr. G. F. L. Elliott (Illawarra Suburbs), Dr. L. Cowlishaw (Kuring-gai District), Dr. W. F. L. Liggins (Northern District), Dr. J. R. Ryan (North Eastern), Dr. C. H. Jaede (South Sydney), Dr. E. L. Newman (Warringah District), Dr. Brooke Moore (Western), Dr. M. L. Coutts (Western Suburbs).

Library.

Dr. G. C. Willcocks was again appointed to the position of Honorary Librarian.

The following are the figures for the year:

Visitors to the library	2,637
Books lent to members	371
Journals lent to members	1,997
Books added to the library	115
Journals added to the library ..	14

Donations of books and periodicals were received from the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, Dr. E. P. Blashki, Dr. G. C. Willcocks, Dr. C. M. Edwards, Dr. S. S. Rosebery, Dr. W. C. McClelland, Dr. C. Nigel Smith, Dr. H. C. R. Darling, Dr. M. S. S. Earlam, Dr. A. J. Gibson, Dr. N. R. Wyndham, the Melbourne University, Australian Dental Association (New South Wales Branch), Medico-Legal Society of Victoria, The Section of Surgery, the Section of Pathology and Bacteriology, the Section of Obstetrics and Gynæcology, and the Section of Medicine.

The following additional journals have been purchased: *The Medical Clinics of North America, The Surgical Clinics of North America, American Journal of Cancer, American Journal of Roentgenology and Radium Therapy, Current Researches in Anesthesia and Analgesia, Journal of Allergy, Journal of Pediatrics, Archives of Neurology and Psychiatry, Archives of Surgery, Proceedings of the Society for Experimental Biology and Medicine, Surgery.*

Affiliated Local Associations of Members.

With the approval of both Local Associations concerned, *viz.*, Warringah District Medical Association and Kuring-gai District Medical Association, the Municipality of Hunter's Hill, which previously formed part of the area of the Warringah District Medical Association, was included within the area of the Kuring-gai District Medical Association.

The Balmain District Medical Association was disbanded, the Drummoyne members being attached to the

ATTENDANCES AT COUNCIL AND STANDING COMMITTEE MEETINGS.

	Council.	Committees.				
		Executive and Finance.	Organization and Science.	Medical Politics.	Hospitals.	Ethics.
DR. G. M. BARRON (President-Elect)	14	10	4	10	3	6
DR. GEORGE BELL (Hon. Treasurer and Premises Attorney)	15	12	2	7	2	3
SIR CHARLES BLACKBURN	11	—	—	—	—	6
DR. K. S. M. BROWN	15	—	—	10	—	—
DR. A. J. COLLINS (Hon. Secretary)	13	9	1	9	2	1
DR. A. M. DAVIDSON	13	8	—	—	—	—
DR. L. A. DEY (Past President)[1]	4	2	—	—	—	—
DR. B. T. EDYE (President)	15	10	4	8	2	3
DR. H. R. R. GRIEVE	14	4	—	7	—	—
DR. P. L. HIPSLEY	15	—	—	8	2	—
PROFESSOR W. K. INGLIS	13	—	5	—	—	—
DR. A. A. PALMER	15	—	—	—	—	6
DR. W. F. SIMMONS	14	—	—	10	—	—
DR. E. H. M. STEPHEN	14	—	—	—	—	6
DR. A. C. THOMAS	13	—	2	—	3	—
DR. E. A. TIVEY	13	—	—	9	—	6
DR. R. C. TRAILL	12	—	—	8	3	—
DR. W. VICKERS	13	9	—	—	3	—
DR. A. S. WALKER	12	—	2	—	—	3
DR. G. C. WILLCOCKS (Hon. Librarian)	12	11	—	—	—	—
Meetings held	15	12	5	11	3	6

[1] Leave of absence April-December, 1938.

Western Suburbs Medical Association and the Balmain members to the South Sydney Medical Association with the approval of the Local Associations concerned.

Border (affiliated 1908): *Honorary Secretary*, Dr. L. S. Woods. Membership, 13. Two meetings were held.

Canterbury-Bankstown (affiliated 1930): *Chairman*, Dr. A. R. Hudson; *Honorary Secretary*, Dr. G. Russell. Membership, 25. Six meetings were held.

Central Northern (affiliated 1910): *Honorary Secretary*, Dr. A. C. Arnold. Membership, 80.

Central Southern (affiliated 1909): *Chairman*, Dr. G. H. Kennett; *Honorary Secretary*, Dr. R. G. Woods. Membership, 29. Two meetings were held.

Central Western (affiliated 1910): *Chairman*, Dr. A. L. Bryant; *Honorary Secretary*, Dr. K. S. M. Brown. Membership, 53. Five meetings were held.

City (affiliated 1918): *Chairman*, Dr. H. A. Ridler; *Honorary Secretary*, Dr. L. R. Flynn. Membership, 32. One meeting was held.

Eastern Suburbs (affiliated 1911): *Chairman*, Dr. C. A. McHardy; *Honorary Secretary*, Dr. C. M. Burns. Membership, 109. Eight meetings were held.

Far South Coast and Tablelands (affiliated 1935): *Chairman*, Dr. J. McKee; *Honorary Secretary*, Dr. K. S. Jones. Membership, 10. Two meetings were held.

Illawarra Suburbs (affiliated 1913): *Chairman*, Dr. T. M. Greenaway; *Honorary Secretary*, Dr. K. S. Richardson. Membership, 42. Eight meetings were held.

Kuring-gai District (affiliated 1929): *Chairman*, Dr. F. A. E. Lawes; *Honorary Secretary*, Dr. H. R. Scrivener. Membership, 64. Four meetings were held.

North Eastern (affiliated 1913): *Chairman*, Dr. N. St. C. Mulhearn; *Honorary Secretary*, Dr. J. B. Oakeshott. Membership, 41.

Northern District (affiliated 1911): *Chairman*, Dr. J. K. Harbison; *Honorary Secretary*, Dr. R. J. Jackson. Membership, 73. Four meetings were held.

South Eastern (affiliated 1914): *Honorary Secretary*, Dr. A. L. Caselberg.

South Sydney (affiliated 1909): *Chairman*, Dr. E. P. Armstrong; *Honorary Secretary*, Dr. C. H. Jaede. Membership, 33. Four meetings were held.

Southern District (affiliated 1909): *Chairman*, Dr. H. O. Lethbridge; *Honorary Secretary*, Dr. R. E. R. Skinner. Membership, 52. Four meetings were held.

Warringah District (affiliated 1929): *Chairman*, Dr. J. S. F. Elphinstone; *Honorary Secretary*, Dr. E. L. Newman. Membership, 57. Seven meetings were held.

Western (affiliated 1908): *Chairman*, Dr. R. D. Mulvey; *Honorary Secretary*, Dr. S. R. Dawes. Membership, 83. Five meetings were held.

Western Suburbs (affiliated 1908): *Chairman*, Dr. R. V. Graham; *Honorary Secretary*, Dr. H. M. Rennie. Membership, 65. Nine meetings were held.

Annual Meeting of Delegates.

The twenty-sixth annual meeting of delegates of the affiliated Local Associations of members with the Council was held on Friday, September 30, 1938. An account of the proceedings of the meeting appeared in The Medical Journal of Australia of October 22, 1938, pages 702-706.

The delegates present at the meeting were as follows: *Border*, Dr. R. A. Robertson; *Canterbury-Bankstown*, Dr. L. Abramovich; *Central Northern*, Dr. A. T. Roberts; *Central Southern*, Dr. R. O. Williams; *Central Western*, Dr. G. N. M. Aitkens; *City*, Dr. A. M. Gledden; *Eastern Suburbs*, Dr. B. W. Stevenson; *Illawarra Suburbs*, Dr. G. F. L. Elliott; *Kuring-gai District*, Dr. W. L. Kirkwood; *Northern District*, Dr. R. J. Jackson; *North Eastern*, Dr. J. R. Ryan; *Southern District*, Dr. R. E. R. Skinner; *South Eastern*, Dr. A. L. Caselberg; *South Sydney*, Dr. G. Cummins; *Western*, Dr. K. C. T. Rawle; *Western Suburbs*, Dr. R. F. Back; *Warringah District*, Dr. R. V. Bretherton.

Sections for the Study of Special Branches of Medical Knowledge.

Anæsthesia (inaugurated 1934): *Honorary Secretary*, Dr. H. J. Daly.

Genito-Urinary and Venereal Diseases (inaugurated 1928).

Hygiene and Preventive Medicine (inaugurated 1922).

Medical Literature and History (inaugurated 1925): *Honorary Secretary*, Dr. L. Cowlishaw.

Medicine (inaugurated 1924): *Chairman*, Dr. H. J. Ritchie; *Honorary Secretary*, Dr. K. B. Noad. Three meetings were held in conjunction with meetings of the Branch.

Neurology and Psychiatry (inaugurated 1924): *Chairman*, Dr. G. P. U. Prior; *Honorary Medical Secretary*, Dr. D. W. H. Arnott; *Honorary Secretary*, Dr. J. A. H. McGeorge. Two meetings were held in conjunction with meetings of the Branch.

Obstetrics and Gynæcology (inaugurated 1925): *Chairman*, Dame Constance D'Arcy; *Honorary Secretary*, Dr. G. G. L. Stening. Five meetings were held, two in conjunction with meetings of the Branch.

Orthopædics (inaugurated 1923): *Chairman*, Dr. Keith Smith; *Honorary Secretary*, Dr. W. L. Macdonald. Two meetings were held in conjunction with meetings of the Branch.

Oto-Rhino-Laryngology (inaugurated 1924): *Chairman*, Dr. H. S. Marsh; *Honorary Secretary*, Dr. E. P. Blashki. One meeting was held in conjunction with a meeting of the Branch.

Pædiatrics (inaugurated 1921): *Chairman*, Dr. D. G. Vickery; *Honorary Secretary*, Dr. W. P. MacCallum. Two meetings were held in conjunction with meetings of the Branch.

Pathology and Bacteriology (inaugurated 1924): *Chairman*, Dr. O. Latham; *Honorary Secretary*, Dr. F. S. Hansman. Two meetings were held in conjunction with meetings of the Branch.

Radiology (inaugurated 1926): *Chairman*, Dr. A. T. Nisbet; *Honorary Secretary*, Dr. H. M. Cutler. Two meetings were held in conjunction with meetings of the Branch.

Surgery (inaugurated 1925): *Chairman*, Dr. J. C. Storey; *Honorary Secretary*, Dr. C. E. Winston. Two meetings were held in conjunction with meetings of the Branch.

British Medical Association Lectures.

Lectures were arranged as follows:

Northern District Medical Association: Tamworth, September 21, 1938, Dr. B. T. Edye, "Acute Abdominal Emergencies"; Armidale, January 30, 1939, Dr. C. G. McDonald, "Nephritis".

North Eastern Medical Association, Lismore, April 2, 1938: Dr. I. D. Miller, "Surgical Aspects of Intracranial Emergencies".

Federal Council.

The Federal Council of the British Medical Association in Australia met in Sydney on June 11 and 12, December 12 and 13, 1938, and in Melbourne on June 30, 1938, and March 14, 15, 16, 18 and 19, 1939, Dr. George Bell and Dr. W. F. Simmons attending as representatives of the Branch.

Dr. George Bell, Dr. W. F. Simmons, Dr. H. R. R. Grieve and Dr. R. C. Traill were the representatives of the Branch on the Federal National Health Insurance Committee, which was appointed by the Federal Council to deal with all matters relating to national health insurance.

Australasian Medical Congress (British Medical Association).

The Sixth Session of the Australasian Medical Congress (British Medical Association) will be held in Perth, Western Australia, on August 31 to September 7, 1940, under the Presidency of Dr. D. D. Paton.

Dr. W. Vickers has been appointed Honorary Local Secretary.

National Health Insurance.

Much attention has been given to this all-important subject during the year.

Eight special meetings of the Council were convened to discuss various aspects of the question.

In order to assist the Council, a Special Committee consisting of seven members of Council and nine members, representing metropolitan and country general practitioners, was constituted. Five meetings of the Committee were held.

In addition, two general meetings of the Association were held to consider the proposed terms of service of medical practitioners.

The representatives of the Branch on the Federal National Health Insurance Committee conveyed to that body the many objections of members to the *National Health and Pensions Insurance Act*, 1938.

To the Local Associations and especially to the representatives of these bodies, who at the invitation of the Council attended meetings, the Council is grateful for their loyal cooperation and support.

To those members who gave evidence before the Royal Commission, the Council is also grateful.

Special meetings of the Association will be held at an early date to consider the latest proposals of the Commonwealth Government.

Contract Practice.

1. The approved Common Form of Agreement for use between medical officer and friendly society lodge continues to work satisfactorily, and the medical service constituted by it is of the greatest value to the community.

The number of members who undertake friendly society lodge practice is 728.

2. At the request of the Australian Natives' Association, the question was reopened of extending recognition to it in connexion with contract medical benefit attendance on its members. It was decided that there should be no alteration in the existing relations with the Australian Natives' Association.

3. A proposal for a national medical service—general practitioner—on a contributory basis has been submitted to Local Associations for their consideration.

Hospitals.

1. Gloucester House, Royal Prince Alfred Hospital. The Board, Royal Prince Alfred Hospital, was requested to review at a future date its decision in regard to the attendance at Gloucester House of medical practitioners other than those on the honorary medical staff of the hospital.

2. Royal North Shore Hospital. Termination of appointments of twelve members of the honorary medical staff.

Strong exception has been taken to the manner in which the appointments of twelve members of the honorary medical staff, Royal North Shore Hospital, were terminated, and representations in regard thereto have been made to the Minister for Health, the Hospitals Commission of New South Wales and the Board of the Hospital.

It has been pointed out to the responsible authorities that, in the interests of the public and the efficiency of the hospital, continuity of tenure of office as a member of the honorary medical staff is essential, and that, unless the profession can be guaranteed such continuity, serious consideration would have to be given to the question of terminating honorary service.

3. Recent Graduates—Resident Medical Officer Appointments. Representations were again made this year to the Hospitals Commission of New South Wales that, in the public interest, a position as resident medical officer should be made available to every recent graduate.

As a result of these representations, positions as resident medical officers were found for a number of the recent graduates who had failed to secure appointments in the ordinary way.

Articles and By-Laws.

1. In pursuance of a special resolution which was passed at an extraordinary meeting of the Branch held on April 28, 1938, a new Article (Article 67) dealing with the formation of Special Groups was constituted.

2. In accordance with Article 68 of the Articles of Association, additions to and amendments of the By-Laws were made as follows:

(*a*) A By-Law dealing with the formation of Special Groups (Sections) and replacing the previously existing By-Law 27.

(*b*) A new By-Law (By-Law 21A): "No member shall accept in relation to his professional services any cash order, voucher or other document issued by any business which provides credit for the payment of medical fees, and no member shall have any pecuniary interest in any such business."

The Medical Practitioners' Act.

An Act of Parliament of special importance to medical practitioners, *viz.*, the *Medical Practitioners' Act* of New South Wales, was passed during the year.

It is to be regretted that amendments, which were submitted in the public interest to the responsible authorities by the Council during the passage of the bill, were not accepted.

Dr. J. R. Ryan, of Lismore, has been appointed, on the nomination of the Council, as provided for in Section 5 (3) of the Act, a member of the Medical Board of New South Wales.

British Medical Agency of New South Wales Limited.

The annual meeting of members of the Agency was held at British Medical Association House, 135, Macquarie Street, Sydney, on Tuesday, August 9, 1938.

In presenting the report of the Directors on the business of the Agency for the year ended June 30, 1938, the Chairman, Sir Robert Wade, explained that the success which had been experienced during that year was due very largely to the operations of Medical Finance Limited, as the result of which many sales of practices had been made.

During the year under review considerable increase had taken place in income earned from other sources, chiefly in the direction of commissions on new business written with the Australian Mutual Provident Society, and in renewal commission on policies of members on the Agency Register.

The net profit for the year, after providing for a reserve to cover taxation, was over £1,500.

The report of the directors and the auditors' report was adopted and the retiring directors, Sir Robert Wade (chairman), Dr. F. Brown Craig, Dr. A. M. Davidson and Dr. George Bell, were reelected for the ensuing year.

In his report the chairman appealed to all members of the Association to give to the Agency their active support.

Medical Finance Limited.

At the meeting of shareholders of Medical Finance Limited, held on August 9, 1938, the first annual report of the company was submitted. This showed that the total value of loans granted to members of the profession was approximately £34,000, some of which had already been repaid before the end of the financial year.

The experience of the company over the first twelve months, therefore, clearly indicated the need which previously existed, for a finance company, owned and controlled by the members themselves.

Practically the entire capital was absorbed by the loan requirements during the first twelve months.

Consequently at a meeting of the company, held subsequent to the annual meeting, it was decided to increase the capital of the company in order that sufficient additional capital would be made available to the company.

The retiring directors, Sir Robert Wade (chairman), Dr. F. Brown Craig, Dr. A. M. Davidson, Dr. George Bell and Dr. L. A. Dey, were reelected.

Congratulations.

Congratulations were extended to Sir Robert Wade on the honour conferred on him by His Majesty the King.

Social.

Golf.—The annual competition—18 holes stroke handicap for the H. Rutherford Darling Cup—which was played on the links of the Concord Golf Club on December 1, 1938, was won by Dr. W. P. MacCallum with Dr. A. M. Ping and Dr. R. B. Holliday runners-up.

Premises Revenue Account.

The Premises Revenue Account discloses a net surplus of £3,778 7s. 10d. as against a net surplus of £2,890 8s. 11d. for the year ended December 31, 1937, being an improvement of £887 18s. 11d. This improvement of £887 18s. 11d. in the net amount of revenue earned is mainly due to an increase of £991 15s. 2d. in rent revenue earned, a saving of £149 7s. 2d. in interest paid to the Australian Mutual Provident Society and on debentures, less sundry increases in wages and other expenses.

A comparison of the annual percentages of expenditure to rent revenue from the time of opening the building in 1930 up to December 31, 1938, is as follows:

	Percentage of Expenses to Revenue.	Percentage of Result to Revenue.
½ year to December 31, 1930	119·4	Deficiency 19·4
1 year to December 31, 1931	114·8	Deficiency 14·8
1 year to December 31, 1932	109·8	Deficiency 9·8
1 year to December 31, 1933	97·9	Surplus 2·1
1 year to December 31, 1934	77·7	Surplus 22·3
1 year to December 31, 1935 (including depreciation)	102·1	Deficiency 2·1
1 year to December 31, 1936 (including depreciation)	89·0	Surplus 11·0
1 year to December 31, 1937 (including depreciation)	82·7	Surplus 17·3
1 year to December 31, 1938 (including depreciation)	78·6	Surplus 21·4

The percentages of rent revenue, expenses and depreciation and the percentage of net surplus for the year to the capital value of the British Medical Association House as shown by the books at December 31, 1938, namely, £175,228 12s. 3d., with the previous year's percentages in parentheses, are as follows:

		1937.
Rent Revenue (including amount charged for British Medical Association Branch offices *et cetera*)	10·09%	(9·37%)
Sundry Expenses, Interest and provision for painting building	6·33%	(6·18%)
Depreciation of Building	1·60%	
	7·93%	(1·57%)
Net Surplus for Year	2·16%	(1·62%)

Financial Statement.

The Council has pleasure in presenting to members the balance sheet and accounts in respect of the financial year, which terminated on December 31, 1938.

The net surplus of revenue over expenditure for the year amounted to £4,519 16s. 6d., after making provision for all known expenses.

The sum of £2,951 7s. 4d. has been written off for depreciation of the building (British Medical Association House), office furniture and equipment and the library.

A sum of £200 has been provided out of the current years' revenue to create a reserve for painting the exterior of the building. This amount, for the time being, is used in the business of the Association.

Financial Statements.

Dr. George Bell moved that the statement of receipts and expenditure be received. The motion was seconded by Dr. A. M. Davidson and carried. Dr. Bell also dealt with the balance sheet and financial statement of the premises account. Dr. Davidson seconded Dr. Bell's motion that the statements be received, and the motion was carried.

Branch Account.

Income and Expenditure Account for Year Ended December 31, 1938.

	£ s. d.	£ s. d.
To Salaries	1,808 19 9	
„ Rent—Offices, etc.	1,000 0 0	
„ Printing and Stationery ..	353 11 10	
„ Stamps and Telegrams	216 10 0	
„ Telephones	91 15 1	
„ Legal Expenses	16 16 2	
„ Travelling Expenses	14 4 4	
„ Insurance	5 4 8	
„ Exchange and Bank Charges	7 13 4	
„ Refreshments—Meetings ..	17 19 6	
„ Newspapers	5 5 0	
„ Sundry Petty Expenses	26 18 4	
„ Gratuity	60 0 0	
„ Tea Money	18 15 0	
„ Federal Council	174 2 0	
		3,817 15 0
„ Depreciation—		
Library	103 1 8	
Office Furniture and Equipment	48 5 8	
		151 7 4
„ Balance—Surplus for the year ended December 31, 1938, transferred to Accumulated Funds Account		741 8 8
		£4,710 11 0

	£ s. d.	£ s. d.
By Subscriptions received—		
1938	8,264 9 6	
1937	229 19 0	
Previous years	115 10 0	
		8,609 18 6
Less Proportion due to—		
British Medical Association	2,239 10 6	
The Medical Journal of Australia	1,755 10 0	
		3,995 0 6
		4,614 18 0
„ Interest	4 0 0	
„ Rent Assembly Hall	82 16 0	
„ Sales—C.F.A., etc.	8 17 0	
		95 13 0
		£4,710 11 0

NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION.

Balance Sheet as at December 31, 1938.

	£ s. d.	£ s. d.
FIXED LIABILITIES.		
Debentures—		
136 4% Series "A" at £10 each	1,360 0 0	
431 4·65% Series "B" at £50 each	21,550 0 0	
246 4·65% Series "C" at £10 each	2,460 0 0	
	25,370 0 0	
Less Amount Unpaid	62 0 0	
	25,308 0 0	
Australian Mutual Provident Society (secured by mortgage over property—B.M.A. House)	88,000 0 0	
		113,308 0 0
CURRENT LIABILITIES.		
Sundry Creditors	361 4 6	
Interest Accrued on Mortgage	330 0 0	
Deposit at Call	700 0 0	
		1,391 4 6
OTHER CREDIT BALANCES.		
Subscriptions Paid in Advance		48 6 0
ACCUMULATED FUNDS.		
Balance at December 31, 1937	63,930 12 10	
Add Surplus for year ended December 31, 1938—		
Branch Account £741 8 8		
Premises Account 3,778 7 10		
	4,519 16 6	
	68,450 9 4	
Provision for Painting of Building	600 0 0	
		69,050 9 4
		£183,797 19 10

	£ s. d.	£ s. d.
FIXED ASSETS.		
Land and Building—		
B.M.A. House at cost less depreciation on building	175,228 12 3	
Library at valuation less depreciation	1,075 1 4	
Office Furniture and Equipment at valuation less depreciation	455 12 4	
Debentures in Other Companies—		
Australasian Medical Publishing Co., Ltd. (face value)	100 0 0	
		176,859 5 11
CURRENT ASSETS.		
Sundry Debtors after making provision for doubtful debts—		
Sundry Tenants, Rent, etc.	2,600 5 5	
Sundry Accounts	45 8 4	
		2,645 13 9
Cash on Hand		18 19 0
Commercial Banking Co. of Sydney Ltd.—		
Premises and Branch Current Accounts	1,147 15 6	
Debenture Interest Account	3 5 6	
Fixed Deposits	2,500 0 0	
		3,651 1 0
OTHER DEBIT BALANCES.		
Prepaid Insurance, Rates, etc.		623 0 2
		£183,797 19 10

Sydney, February 8, 1939.

We have examined the foregoing Balance Sheet with the Books of Account of the New South Wales Branch of the British Medical Association, and, having obtained all the information and explanations we have required, we are of the opinion that such Balance Sheet is properly drawn up so as to exhibit a true and correct view of the state of the Company's affairs according to the best of our information and the explanations given to us and as shown by the Books of the Company. In our opinion the Register of Members and other records which the Company is required to keep by law or by its Articles have been properly kept.

F. W. DUESBURY, HARTIGAN & Co.,
Chartered Accountants (Aust.).

B. T. EDYE, President.
GEORGE BELL, Hon. Treasurer.
R. J. STIFFE, F.C.A. (Aust.), Financial Secretary.

London Bank Chambers,
18-20 Martin Place, Sydney.

ELECTION OF OFFICE-BEARERS.

Dr. Edye announced that the following had been elected members of the council for the ensuing year: Dr. George Bell, Sir Charles Blackburn, Dr. K. S. M. Brown, Dr. A. J. Collins, Dr. A. M. Davidson, Dr. L. A. Dey, Dr. H. R. R. Grieve, Dr. P. L. Hipsley, Professor W. K. Inglis, Dr. H. H. Jamieson, Dr. A. M. McIntosh, Dr. W. F. Simmons, Dr. E. H. M. Stephen, Dr. A. C. Thomas, Dr. E. A. Tivey, Dr. R. C. Traill, Dr. A. S. Walker.

Dr. Edye said that Dr. J. W. Hoets and Dr. K. C. T. Rawle had obtained an equal number of votes. The rules provided that in such circumstances the method of deciding an election should rest with the President. He thought that the matter might be determined by lot. Dr. Hoets and Dr. Rawle agreed with this suggestion and Dr. Rawle's name was drawn; he was declared elected.

Messrs. F. W. Duesbury, Hartigan and Company were appointed auditors for the ensuing year.

On the motion of Dr. A. J. Collins, seconded by Dr. K. S. M. Brown, Dr. A. A. Palmer was appointed representative and Dr. W. C. B. Harvey deputy representative of the Branch on the Representative Body, 1939-1940.

On the motion of Dr. W. F. Simmons, seconded by Dr. A. C. Thomas, Dr. W. C. B. Harvey and Dr. H. V. P. Conrick were appointed delegates to attend the 107th annual meeting of the Association, to be held at Aberdeen, July 25 to 28, 1939.

Dr. L. A. Dey moved:

> That Dr. A. A. Palmer, a member of council March 27, 1908, to March 29, 1912, and March 31, 1916, to date, President 1918-1919, Honorary Secretary in 1909, and on several other occasions during the absence of the late Dr. R. H. Todd and Honorary Secretary to two Australasian medical congresses, in 1911 and 1929, be elected a Vice-President of the Association.

The motion was seconded by Dr. George Bell and carried unanimously.

Incoming President's Address.

Dr. George Barron delivered his president's address (see page 565). A vote of thanks was passed to Dr. Barron on the motion of Dr. A. A. Palmer, seconded by Dr. H. R. R. Grieve.

Induction of President.

Dr. B. T. Edye inducted the President for the year 1939-1940 (Dr. George Barron). In doing so he said that in his opinion the activities of the previous year, which had been most strenuous, had brought the profession together, and that the profession was now more solid than it had been in the past. Dr. Edye expressed his keen appreciation of the work of the council and also of those who had been coopted in connexion with national health insurance. These members had always attended willingly and, he was sure, often with great personal inconvenience. The Branch was greatly indebted to them. Dr. Edye wished to thank those members of the Branch who had given evidence before the Royal Commission; he was also most grateful to the Honorary Secretary and to every member of the office staff, including Dr. Hugh Hunter, for the untiring energy and careful attention to detail which they had displayed. He exhorted members of the Branch to place trust in their elected representatives; the events of the previous year had surely taught Branch members to elect their representatives and to stick to them. The council would always welcome constructive criticism, but criticism which was purely destructive was not only useless but disheartening. Dr. Edye concluded by saying that he was sure members would give to their new President, Dr. Barron, their whole-hearted support.

Dr. Barron thanked the members for his election.

VICTORIAN BRANCH NEWS.

The following items of news of interest to members of the Victorian Branch of the British Medical Association are published at the request of the Council of the Branch.

Suburban Out-Patient Dispensaries.

In pursuance of the expressed policy of the Charities Board of Victoria, out-patient dispensaries have now been established in Richmond, Collingwood and Footscray, and it is understood that more are contemplated.

While recognising that a policy covering existing conditions would not be applicable in the event of the introduction of some scheme of national health insurance, the Branch Council has adopted the following points as a basis for the formation of a policy in regard to such institutions.

1. Suburban dispensaries should not be used as special diagnostic centres for radiological and pathological investigations, and patients requiring such special investigations should be referred to existing public hospitals.

2. The function of a suburban dispensary should be the treatment of patients of proved indigency residing in the district, invalid and old age pensioners and sustenance workers, and those referred from public hospitals.

3. No facilities for surgery, other than those required for first aid work and dressings, should be provided at a dispensary.

4. Persons for whose treatment third parties are responsible, such as those coming under the provisions of the *Workers' Compensation Acts*, should not be eligible for treatment at a dispensary, except in cases of extreme emergency, and then only for appropriate initial treatment.

5. The rate of remuneration of a member of the medical staff of a dispensary should be at the rate of one guinea per hour, and attendances should be so regulated as not to require a medical officer to examine or treat an average of more than eight patients per hour.

6. Local members of the British Medical Association should be represented on the committee of management of a dispensary.

7. Appointments of medical officers, under terms and conditions approved by the Branch council, should be made by dispensary committees after applications have been invited by advertisement in the daily Press, and the Branch council is prepared to render assistance, in an advisory capacity, in the selection of medical practitioners seeking dispensary appointments.

Policy of the Council of the Victorian Branch, British Medical Association, in re Workers' Compensation Cases.

1. Fees payable to practitioners for treatment of insured persons shall be in accordance with the British Medical Association scale of fees for private patients. The minimum fee for any one attendance shall be 10s. 6d.

2. Every insured person shall have a free choice of the practitioner who is to carry out his treatment.

3. No practitioner shall enter into an agreement with an insurance company to treat insured patients for whom the company is responsible, unless such agreement is approved by the council and does not in any way interfere with the insured person's freedom of choice of doctor.

4. An insurance company may at any time request the practitioner in charge of an insured person to meet in consultation a medical practitioner employed by the company, and it shall be the duty of the practitioner to accede to such request; provided that the medical officer of the company shall in no way alter or otherwise interfere with the treatment of the insured person without the knowledge and consent of the practitioner.

5. In the event of a disagreement arising between the practitioner in charge of the treatment of an insured person and the medical officer of the insurance company concerned, as to the appropriate treatment of the insured person, both parties shall agree to refer the case to a third practitioner, whose decision shall be final; provided that: (*a*) such practitioner shall be mutually acceptable to both parties; (*b*) such practitioner shall be possessed of recognized qualifications, which render him specially qualified to deal with the case at issue.

6. No insured person shall be eligible for treatment at a public hospital except: (*a*) in case of extreme emergency; (*b*) where it is impossible to obtain the appropriate treatment from a private practitioner.

7. In the event of an insured person being admitted to a public hospital under any of the terms of paragraph 6, the bed which he occupies shall forthwith be declared a pay-bed by the hospital authorities.

8. In the event of the fees due to the hospital authorities for maintenance, and to the practitioner in charge for professional services in respect of an insured person, representing a sum greater than that for which the insurance company is liable, the sum payable by the insurance company in respect of these services shall be divided between them in a proportion to be arranged between the authorities and the medical staff of the hospital concerned.

Based on the above principles a detailed scheme has been evolved and submitted for consideration of the members of the honorary medical staffs of the public hospitals concerned. The scheme is designed to remove patients suffering from industrial injuries from the care of the managements of the public hospitals throughout the State.

Research.

SECOND INTERNATIONAL STANDARD VITAMIN B_1.

The following statement is published at the request of the Director-General of Health, Commonwealth Department of Health.

The standard adsorption product of vitamin B_1, which has hitherto served as the international standard for vitamin B_1, has now been replaced by a preparation of pure crystalline vitamin B_1 hydrochloride (aneurin chloride hydrochloride, or thiamin chloride hydrochloride), and the international unit of vitamin B_1 has been defined as the antineuritic activity of three microgrammes of the new international standard preparation.

A limited quantity of this new international standard has been received at the Commonwealth Serum Laboratories, which laboratories act as the national distributing centre for Australia.

Supplies can be obtained by application to the director. Applicants are reminded, however, that it is not intended that the international standard should be used for all routine tests for vitamin B_1 activity carried out in laboratories or manufacturing establishments.

It is clear that, if used for routine testing, the standard would quickly become exhausted. It is recommended, therefore, that each laboratory carrying out many routine tests for vitamin B_1 activity should be encouraged to establish a local laboratory standard and should assay the potency of the local standard by means of a sample of the international standard supplied for the purpose, and the international standard should be used subsequently only for the periodical checking of the local laboratory standards.

University Intelligence.

THE UNIVERSITY OF SYDNEY.

A MEETING of the Senate of the University of Sydney was held on Monday, April 3.

Dr. Arthur E. Mills was reelected to the position of Deputy Chancellor for the year 1939.

Mr. B. C. Harkness, Acting Director of Education, tendered his resignation as a Fellow of the Senate as from April 5. It is understood that his successor will be Mr. G. Ross Thomas, B.A., Director of Education, who returns to Sydney this month.

The Dr. John Osborne R.N. Scholarship has been awarded to Mr. E. J. Halliday, a first-year student in the Faculty of Medicine. Mr. Halliday gained the highest marks of all candidates at the Leaving Certificate Examination held in November last. This scholarship is tenable by a student of Saint Paul's College, and the stipend is £50 per annum for two years.

Appointments to the teaching staff: Mr. J. W. Evans as Junior House Surgeon in the Faculty of Veterinary Science; Miss Kathleen Winning, M.B., Ch.M., D.C.H., as Lecturer in Mothercraft in the room of Miss Margaret Harper (resigned); Miss Edith Gardner, B.Sc., as Demonstrator in Biochemistry.

Mr. D. G. Salier has been appointed as Temporary Lecturer in Physics.

The following degree was conferred: Master of Surgery (Ch.M.), John Leslie Dart.

Proceedings of the Australian Medical Boards.

TASMANIA.

THE undermentioned has been registered, pursuant to the provisions of the *Medical Act*, 1918, of Tasmania, as a duly qualified medical practitioner:

Holt, Ian Wellesley, M.B., B.S., 1939 (Univ. Sydney), Devon Hospital, Latrobe.

Books Received.

THE MEDICAL PRESS AND CIRCULAR, 1839-1939. A HUNDRED YEARS IN THE LIFE OF A MEDICAL JOURNAL, by R. J. Rowlette, M.D., F.R.C.P.I.; 1939. London: The Medical Press and Circular. Crown 4to, pp. 127, with illustrations.

FOOD, HEALTH, VITAMINS, by R. H. A. Plimmer, D.Sc., and V. G. Plimmer; Eighth Edition; 1939. London: Longmans, Green and Company Limited. Crown 8vo, pp. 243, with coloured frontispiece and diagrams in the text. Price: 5s. net.

BACTERIAL METABOLISM, by M. Stephenson, Sc.D.; Second Edition; 1939. London: Longmans, Green and Company. Demy 8vo, pp. 405, with illustrations. Price: 21s. net.

DIE ENDOKRINEN DRUSEN DES GEHIRNS EPYPHYSE UN HYPOPHYSE EIN BLICK IN EIN INTERESSANTES GEBIET, by P. Niehans, M.D.; 1938. Bern: Hans Huber. Super royal 8vo, pp. 280. Price: Schw. Fr. 10·50.

UBER DIE INTEGRATIVE NATUR DER NORMALEN HARNBILDUNG, by G. Ekehorn, D.M.; Volumes I, II and III; 1938. Helsingfors: Mercators Tryckeri. Medium 8vo, pp. 1431.

RESEARCH IN MEDICINE AND OTHER ADDRESSES, by T. Lewis, C.B.E., F.R.S., F.R.S.E., M.D., D.Sc., LL.D., F.R.C.P.; 1939. London: H. K. Lewis and Company Limited. Demy 8vo, pp. 82. Price: 5s. net.

LEAGUE OF NATIONS PUBLICATIONS. SURVEY OF NATIONAL NUTRITION POLICIES, 1937-1938; 1938. Geneva: League of Nations Publications Department; Australia: H. A. Goddard. Royal 8vo, pp. 120. Price: 2s. 6d. net.

THE SCIENCE AND ART OF JOINT MANIPULATION, by J. Mennell, M.A., M.D., B.C.; Volume I: The Extremities; 1939. London: J. and A. Churchill Limited. Crown 4to, pp. 245, with illustrations. Price: 18s. net.

A HANDBOOK OF OPHTHALMOLOGY, by H. Neame, F.R.C.S., and F. A. Williamson-Noble, F.R.C.S.; Third Edition; 1939. London: J. and A. Churchill Limited. Medium 8vo, pp. 346, with 12 plates, containing 46 coloured illustrations and 143 text figures. Price: 13s. 6d. net.

CLINICAL BACTERIOLOGY, by F. A. Knott, M.D., M.R.C.P., D.P.H.; 1939. London: J. and A. Churchill Limited. Demy 8vo, pp. 432, with 60 illustrations, including 12 plates. Price: 12s. 6d. net.

GARDINER'S HANDBOOK OF SKIN DISEASES, revised by J. Kinnear, T.D., M.D., M.R.C.P.; Fourth Edition; 1939. Edinburgh: E. and S. Livingstone. Crown 8vo, pp. 255, with illustrations, including 16 coloured plates. Price: 10s. 6d. net.

TEXTBOOK OF MEDICINE, by various authors, edited by J. J. Conybeare, M.C., D.M., F.R.C.P.; Fourth Edition; 1939. Edinburgh: E. and S. Livingstone. Demy 8vo, pp. 132, with illustrations. Price: 21s. net.

THE BASIC MECHANICS OF HUMAN VISION, by R. Brooks Simpkins; 1939. London: Chapman and Hall Limited. Demy 8vo, pp. 236, with illustrations. Price: 12s. 6d. net.

DIABETIC DIETS, by W. W. Ingram, M.C., M.D., F.R.A.C.P.; Second Edition; 1939. Australia: Angus and Robertson Limited. Crown 8vo, pp. 16. Price: 1s. net.

THE DIABETIC LIFE: ITS CONTROL BY DIET AND INSULIN. A CONCISE PRACTICAL MANUAL FOR PRACTITIONERS AND PATIENTS, by R. D. Lawrence, M.A., M.D., F.R.C.P.; Eleventh Edition; 1939. London: J. and A. Churchill Limited. Large crown 8vo, pp. 255, with 18 illustrations. Price: 8s. 6d. net.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Friend, Kenneth James, M.B., B.S., 1939 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Holme, John Leicester, M.B., B.S., 1939 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

The undermentioned has applied for election as a member of the Western Australian Branch of the British Medical Association:

Reynolds, Marjorie, M.B., B.S., 1931 (Univ. Melbourne), Roebourne.

Diary for the Month.

Apr. 18.—New South Wales Branch, B.M.A.: Medical Politics Committee and Ethics Committee.
Apr. 19.—Western Australian Branch, B.M.A.: Branch.
Apr. 20.—New South Wales Branch, B.M.A.: Clinical Meeting.
Apr. 26.—Victorian Branch, B.M.A.: Council.
Apr. 27.—New South Wales Branch, B.M.A.: Branch.
Apr. 27.—South Australian Branch, B.M.A.: Branch.
Apr. 28.—Queensland Branch, B.M.A.: Council.
May 2.—New South Wales Branch, B.M.A.: Organization and Science Committee.
May 3.—Victorian Branch, B.M.A.: Branch.
May 3.—Western Australian Branch, B.M.A.: Council.
May 4.—South Australian Branch, B.M.A.: Council.
May 5.—Queensland Branch, B.M.A.: Branch.
May 9.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
May 12.—Queensland Branch, B.M.A.: Council.
May 16.—New South Wales Branch, B.M.A.: Ethics Committee.
May 17.—Western Australian Branch, B.M.A.: Branch.
May 18.—New South Wales Branch, B.M.A.: Clinical Meeting.

Medical Appointments.

Dr. L. J. L. Price has been appointed Government Medical Officer at The Rock, New South Wales.

* * *

Dr. C. Y. Bland has been appointed a Medical Officer in the Office of the Director-General of Public Health of New South Wales.

* * *

Dr. B. F. Gates has been appointed a Medical Officer in the Office of the Director-General of Public Health of New South Wales.

* * *

Dr. J. G. Hulme has been appointed Official Visitor to the Willowburn Mental Hospital, pursuant to the provisions of *The Hospitals Act of* 1936 of Queensland.

* * *

Dr. C. T. C. de Crespigny has been appointed Chairman of the Council of the Institute of Medical and Veterinary Science of Adelaide, South Australia.

* * *

Dr. C. Gurner and Dr. B. S. Hanson have been appointed Honorary Deep X Ray Therapist and Honorary Assistant Radiologist, respectively, at the Adelaide Hospital, Adelaide, South Australia.

* * *

Dr. J. A. R. Murphy has been appointed Medical Superintendent of the Waterfall Sanatorium, Office of the Director-General of Public Health in the Department of Public Health of New South Wales.

* * *

Dr. F. W. Arden has been, according to the provisions of *The Cremation Act*, 1913 *to* 1935, of Queensland, issued a licence authorizing him to sign permissions and certificates for cremation and to grant permission to cremate any human body after death.

* * *

Dr. D. M. McWhae and Dr. M. K. Moss have been appointed members of the Board of Visitors to the Claremont, Greenplace and Whitby Falls Mental Hospitals, in pursuance of the provisions of the *Lunacy Act*, 1903-1920, of Western Australia.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xvi to xviii.

Naremben Road Board, Naremben, Western Australia: Medical Officer.
The University of Melbourne, Victoria: Demonstratorships in Clinical Biochemistry.

Medical Appointments: Important Notice.

Medical Practitioners are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
New South Wales: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
Victorian: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
Queensland: Honorary Secretary, B.M.A. House, 235, Wickham Terrace, Brisbane, B.17.	Brisbane Associate Friendly Societies' Medical Institute. Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
South Australian: Secretary, 178, North Terrace, Adelaide.	All Lodge appointments in South Australia. All Contract Practice Appointments in South Australia.
Western Australian: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.

Editorial Notices.

Manuscripts forwarded to the office of this journal cannot under any circumstances be returned. Orginal articles forwarded for publication are understood to be offered to The Medical Journal of Australia alone, unless the contrary be stated.

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